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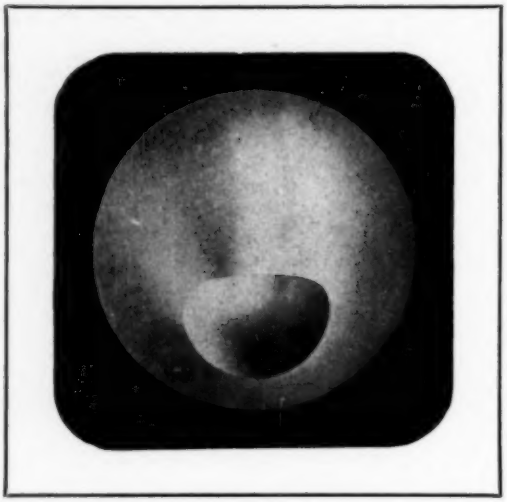

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AMERICAN JOURNAL OF OPHTHALMOLOGY
VOLUME 16 AUGUST, 1933 NUMBER 8

THE CONTINUOUS CIRCULATION OF THE AQUEOUS HUMOR

MANUEL URIBE TRONCOSO, M.D.
NEW YORK CITY

Analysis and continuous critique of the work done by Friedenwald and Pierce on this subject, on the basis of his own results, and a statement of the views held by the followers of the theory of the circulation of the aqueous humor are given.

In the last three decades the ideas in regard to the circulation of the aqueous have undergone a complete change. The old conception of a continuous outflow of fluid through Schlemm's canal and the veins of the iris was shaken to its foundations by the new school of Weiss and Magitot, who consider the aqueous humor a dialysate of the blood plasma and almost stagnant in the anterior chamber, being subject only to changes in pressure and to the laws of osmosis and diffusion.

Being a convinced adherent of the old idea that a constant current flows from the posterior to the anterior chamber and then leaves the eye in part through Schlemm's canal and the episcleral veins, and in part by the veins of the iris, I was very much interested in the two articles published in the *Archives of Ophthalmology* by Drs. Jonas S. Friedenwald and H. T. Pierce on the circulation of the aqueous[1]. By numerous experiments on dogs they have again confirmed the existence of a constant current of fluids within the eye. In their researches they used Leber's filtration manometer in living eyes, and ascertained in two series of experiments: first, the rate of flow, and second, the mechanism of reabsorption of fluid in the anterior chamber. Years ago the writer made a great number of experiments with this instrument but was very much disappointed with the results and, in fact, with the whole of Leber's method of ascertaining the rate of outflow. For this reason it seems advisable to restate the facts on which my conclusions in regard to the inaccuracies and defects of the instrument and Leber's method were founded.

The principle on which Leber's manometer is operated is that when a liquid is injected through a needle into the anterior chamber under a certain pressure, the amount of the inflow in a given time may be measured by the displacement of an air bubble in a horizontal, graduated tube. He believed that the amount and rate of inflow into the anterior chamber also measured the amount and rate of the outflow through Schlemm's canal and the episcleral vessels. When I used the apparatus in a series of experiments[2] to determine the rate of outflow with a saline solution and then with liquids containing albumin in various proportions, I soon discovered that Leber's method is based on an *a priori* assumption, and that it was incorrect to assume that the amount of outflow corresponds to the amount injected into the chamber. To prove my contention I used not only pigs' eyes but also human eyes, enucleated as soon as possible after death. The outer surface of the eye was dried, removing all blood clots and liquids. It was then placed in a porcelain capsule and carefully weighed. The weight of the empty capsule had been previously ascertained. The experiment was started by inserting a Leber needle into the anterior chamber and injecting the fluid under normal intraocular pressure, usually for one-half to one hour. Watching the outside of the eyeball during this whole period, it was observed that liquid continually flowed in tiny droplets through the cut ends of the episcleral vessels around the scleral limbus, and slowly collected at the bottom of the capsule. At the end of the experiment the total amount of the liquid injected was recorded, the eyeball was removed

from the capsule and the latter again carefully weighed. Deducting the weight of the capsule itself the rest represented the real amount of the outflow. Comparing it with the liquid injected, I was able to prove that about one-third, and in some cases even half, of the liquid injected into the anterior chamber had been retained inside of the eyeball. Thus the figures of Leber and Niesnamoff represented only the rate of inflow into the anterior chamber, but not the true amount of outflow, which was much lower. They found a rate of 11 c.mm. per minute in pig eyes and 5.5 c.mm. per minute in man, but in my own experiment the rate of the real outflow was reduced to 2.6 c.mm. per minute in human eyes. Leber immediately disputed the value of my experiments attributing my criticisms and results to errors of technic and not to defects of the apparatus, and a long polemic followed[3]. After an extended discussion and after repeating my own researches Leber admitted at last that I was right. In his experiments he found that not only one half but in some cases even two thirds or three fourths of the liquid injected remained inside of the eye. He persisted however in asserting that my conclusions were not well founded. I proved later that the difference in our figures was due to a faulty graduation of the tube of the instrument which I used, and which was bought from the manufacturer whom Leber had recommended. However as I used only measures in weight for the calculations throughout the whole experiment, my conclusions were correct.

Another important fact which I insisted should be considered in these researches was the difference in pressure in the anterior and posterior segments of the eye during the experiment. As stated before, in Leber and Niesnamoff's and in my own researches, dead eyes of pigs and dead human eyes were used. As they were necessarily soft when the injection was started, the anterior chamber became increasingly deep, according to the condition of the eye; viz., the state of liquefaction of the vitreous and the elasticity of the scleral and inner membranes. Thus the pressure was very different at first in both the anterior and posterior segments of the eye and the liquid retained during the experiment slowly accumulated in the vitreous and distended the sclera, changing its shape to a nearly spherical globe. To eliminate this error I inserted a second needle into the vitreous and connected it with a reservoir filled with normal saline solution, which could be moved to various heights above the eye. When the pressures were equal on both needles the anterior chamber did not increase in depth and the rate of inflow diminished considerably, being about half of the one found when no especial vitreous pressure existed. If the latter was increased gradually the amount of inflow decreased rapidly, until at a pressure of 27 to 31 mm. Hg., all inflow stopped. Reducing the vitreous pressure the inflow started again in proportion to the decrease and the cycle could be reproduced at will. Examining microscopical sections of human eyes frozen during the period of stoppage of the inflow, the angle of the anterior chamber was found to be occluded by the application of the root of the iris against the sclerocornea.

Besides the experiments in dead eyes, Leber and Niesnamoff made a few in living animals. In the rabbit they found that the inflow from the manometer into the anterior chamber was 1 c.mm. per minute, at the intraocular pressure of 25 mm. Hg. The animal was then killed by bleeding and the experiment continued. They then observed, that the rate of inflow had increased to 7 c.mm. per minute. As the secretion of the ciliary body was absent after death, Niesnamoff concluded that this secretion should correspond to the difference between those figures, or 6 c.mm. per minute. This author however did not consider the great role of the diminished vitreous pressure and of the stoppage of the blood circulation, which allows a greater inflow of liquid from the apparatus, even if the intraocular pressure were maintained constant.

On account of the numerous causes of error incident to the use of Leber's manometer, especially the great disproportion between the inflow and the real

amount of the outflow, I entirely abandoned the method and the use of dead eyes and undertook a series of researches on living eyes with a new technic[4]. I chose the rabbit on account of the facility with which the eyeball can be drawn outside the orbit and isolated, so that the amount of outflow can be accurately measured. The experiments were conducted as follows:

After dissecting the conjunctiva all around the limbus, the eyeball is slowly drawn out of the orbit with forceps and then passed through a hole in a rubber-dam sheet which isolates it from the liquids of the conjunctiva and orbit. It is then immersed in a glass cup containing olive oil. In a short time small droplets of sero-sanguineous fluid ooze from the episcleral vessels. If these droplets are removed with a probe, new ones reform slowly. After one hour the eye is withdrawn and the sero-sanguineous liquid that has collected at the bottom of the cup is centrifugalized. The amount of clot and clear liquid thus obtained is compared with the volume of clot and serum in the blood taken from the ear of the animal. It was found that, allowing for the plasma of the blood contained in the outflow, a large quantity of liquid remained, which was undoubtedly aqueous. The rate of flow was calculated at 2 to 3 c.mm. per minute.

Friedenwald and Pierce quoting these experiments make the objection that the intraocular venous pressure must have been increased by the dislocation of the eyeball and the pressure of the lids on the tissues behind the equator. Besides the drainage was measured against zero pressure in the outflow vessels. They believe however that "it is the closest approach to a true measurement of the intraocular circulation," and that "the figures of 2 to 3 c.mm. per minute are not far from the true magnitude."

Reopening the study of this subject Friedenwald and Pierce used a Leber manometer modified by the addition of a second horizontal tube parallel to the graduated tube, and the substitution of the large mercury pump by a reservoir which could be moved to different heights. They made two series of experiments: the first to determine the normal rate of outflow of the aqueous from the anterior chamber in the living eye, and the second to determine the mechanism by which the reabsorption of this fluid takes place.

In the first series, at variance with Leber and other investigators, the authors blocked the channels of excretion of the anterior chamber by the injection of the animal's own serum through a second needle inserted in the anterior chamber. This they found to be the best method of stopping the outflow, after using a considerable variety of colloid solutions and fine-grain suspensions.* By this ingenious method of blocking the angle they could observe that instead of liquid being admitted into the eye from the apparatus, a constant flow of fluid took place from the anterior chamber to the apparatus, when the normal intraocular pressure was not disturbed. The outflow was continuous and uniform at the rate of 1 c.mm. per minute. This the authors submit as proof that there is a constant current of fluid out of the anterior chamber in normal conditions.

This is an important conclusion. Should the aqueous be stagnant and move only by changes in the blood pres-

* It is perhaps interesting to mention that I was probably one of the first to study the effects of albuminous solutions made with animal serum in the blocking of the angle. In a series of experiments[5] made with dog and horse sera, I tried to determine in what proportion the filtration which I called "apparent," that is, the amount of inflow into the anterior chamber as given by Leber's manometer, varied with different concentrations of serum. On account of the great variation of the inflow from one eye to another I measured first the rate of inflow with normal saline solution and then injected into the same eye solutions the albumin content of which varied from 1 percent to, 2, 3, 4, and 6 percent. I found that the rate of inflow was greatly diminished; in human eyes the diminution, taking the rate of saline solution as a unit, varied from two fifths to seven ninths. In one eye, which was left for several hours in connection with Leber's manometer filled with albuminous solution at 6 percent (the eye being placed in a dry capsule and covered with a tin sheet to prevent evaporation), the inflow at the end gradually diminished to 0. Upon raising the pressure from normal to 35, 50, and 75 mm., the inflow started again each time but gradually sank to 0.

sure and intraocular pressure, no uniform current could be observed but only an irregular outflow, with periods of complete stoppage. Duke-Elder, trying to reconcile both theories, states that the conception of a dialysate in equilibrium with the capillary blood and a constant current is tenable if we remember that by the contractions of the muscles in the orbit and of the ciliary muscle there should be a rise in the intraocular pressure, and as the pressure in Schlemm's canal then becomes lower than the intraocular, a current of liquid virtually constant, but minimal, should be established under such constantly changing pressure conditions[5].

However, in Friedenwald and Pierce's experiments the animal was completely anesthetized and no movements of the eyeball or of the ciliary muscle occurred. Still there was a constant current even with complete immobility of the whole body. These authors also support the eclectic theory that the aqueous humor does not simply trickle out of the anterior chamber by hydrostatic pressure, but is reabsorbed into the blood stream on account of the difference in osmotic pressure of the plasma proteins in the veins of the iris and Schlemm's canal and that of the aqueous. These forces are the same that are concerned in dialysis, and therefore, they assert, there should be no difference between the fluid remaining in the anterior chamber if a continuous current exists, or if the aqueous is simply a dialysate from the blood. They claim, as Duke-Elder had stated before, that the pressure in Schlemm's canal and in the veins of the iris, is slightly above the intraocular pressure, and therefore the absorption of the aqueous takes place not by filtration but by the difference of the osmotic pressure in the blood plasma and in the aqueous, which amounts to 20 to 25 mm. Hg. The excess of this difference over the difference in the hydrostatic pressure within and without the canal and veins, which, they state, is probably only 1 or 2 mm. Hg above the intraocular pressure, produces the absorption.

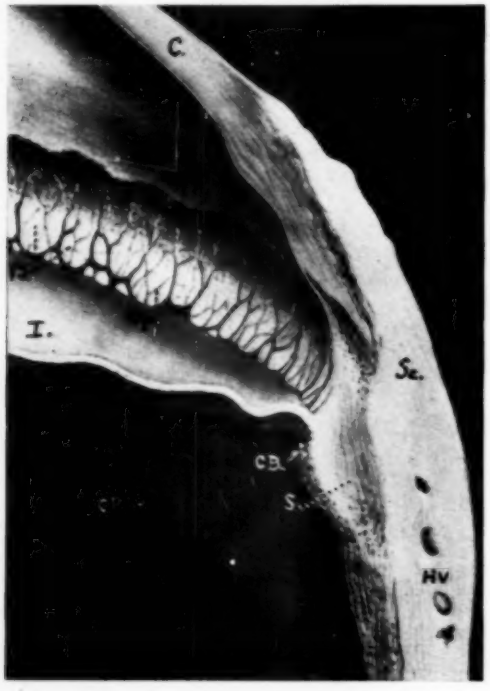

Fig. 1 (Uribe Troncoso). Drawing of a macroscopic section of a dog's eye seen under great magnification. I., iris; C., cornea; Sc., sclera; CB, ciliary body; Lp., ligamentum pectinatum, whose fibers, at different levels, attach the iris to the sclera; FS, Fontana's spaces; S., sinus of the anterior chamber filled with numerous fibers of the pectinate ligament and reaching far backward; Hv, sections of the large veins of Hovius's plexus; CP., ciliary processes.

Friedenwald and Pierce and Duke-Elder, assume that the pressure in Schlemm's canal is higher than the intraocular because of the connections between the canal and the intrascleral veins, where, by direct measurement in dogs' eyes, Duke-Elder found the blood pressure to be a little higher than the intraocular. The writer does not agree with this assumption, and believes that in man the pressure in Schlemm's canal is much lower than that in the veins and probably always lower than the intraocular pressure. Anatomically, the difference in the region of the angle in man and in the dog is very marked. In the latter (fig. 1) there is not a well-differentiated Schlemm's canal. The large veins of Hovius's plexus which run inside the sclera are much larger and more numerous than the intrascleral plexus in man. In some places these veins

come so near the inner wall of the sclera (fig. 2), that some authors (Nuel and Benoit[6]) have asserted that their lumen opens directly into Fontana's spaces, because the injection of India ink always pentrates the veins. This has not been verified by other observers. The fact remains that the intrascleral veins in the

Fig. 2 (Uribe Troncoso). Microscopic section of the angle in the dog. I, iris; L.p., ligamentum pectinatum. The innermost and thicker fibers have been called "suspensor ligament of the iris." S., sinus or recess of the anterior chamber filled with the fibers of the pectinate ligament; F.S., Fontana's spaces between the fibers. The sinus reaches far backward, separating the ciliary body from the sclera. C.M., ciliary muscle; HV, large vein of Hovius's plexus, a smaller branch of which, V', comes near the inner wall of the sclera from which it is separated by some thickness of fibers. From it start fine connecting branches, which almost reach the inner scleral wall.

dog are not comparable to Schlemm's canal in man, which is a specialized organ. The blood pressure in the former is probably higher by 1.5 mm. than the intraocular, as Duke-Elder found. But in man the canal of Schlemm is not in direct, through-and-through communication with the intrascleral veins. It is really a *diverticulum* of the venous circulation, its connections with the veins being made only by narrow, conical channels or "collectors" inserted very obliquely into the canal by an enlarged base, while the apex is connected with the veins. According to Maggiore[7], there are only twenty to thirty such collectors around the limbus (fig. 3). Their lumen

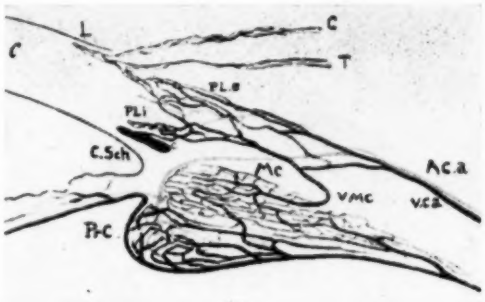

Fig. 3 (Maggiore). Schematic representation of serial sections of an injected human, adult eye. Shows the relation of the different vascular plexus in the angle and surrounding structures. C, conjunctival vessels; T, vessels of Tenon's capsule; PL.e, episcleral plexus; PL.i, intrascleral plexus; C.Sch, Schlemm's canal; c, collector joining the canal with the intrascleral veins; Ac.a, V.ca, anterior ciliary artery and vein; VMc, veins connecting the ciliary muscle plexus with the intrascleral veins; L, limbus; *C*, cornea; PrC, ciliary processes; Mc, ciliary muscle.

is almost virtual being scarcely the size of a red corpuscle. In these conditions the pressure inside the canal must certainly be much lower than in the connecting intrascleral veins, where the circulation flows through and through directly to the outside. Hence a hydrostatic outflow is possible from the anterior chamber to Schlemm's canal. At normal intraocular pressure the absorption of the aqueous is probably made both by osmotic pressure through the veins of the iris and by hydrostatic pressure through the canal. The absorption through the latter, however, should be smaller in amount, as the capacity of the canal is much smaller than the total surface of absorption provided by the veins of the iris. In pathological conditions in man, when the intraocular pressure increases, the excretion of the aqueous through Schlemm's canal must also increase very rapidly and continue even when the veins of the iris become obliterated by compression, because the lumen of the canal and the intrascleral veins are always kept open by the surrounding scleral tissue.

Rate of flow. In the dog Friedenwald and Pierce found the outflow to be 1 c.mm. per minute. This amount is perhaps too small for an animal that has a

comparatively large anterior chamber and a deep sinus recess reaching far back (fig. 1, S.). The small outflow they obtained could be explained as due to a retention of fluid in the eye. The authors assert that they blocked the escape through the angle by the injection of the animal's own serum into the anterior chamber, and to check the thoroughness of the blocking they made a comparative experiment, injecting liquid into the intact eye of the animal and measuring the inflow in both eyes, at "a greater pressure than the normal." They found that the rate of flow was three fourths to nine tenths less in the eye containing the serum than in the other. This, however, is not a direct proof but only an assumption, as in both cases only the inflow at a greater pressure than normal was really measured.

It would have been very interesting had they found, comparatively, the amount of outflow after blocking the angle in other animals, such as the cat, which has a very wide ciliioscleral sinus, and in the rabbit, in which the pectinate ligament is not so large, and in which the ciliioscleral sinus is not so deep as in the dog.

In their second series of experiments Friedenwald and Pierce used Leber's method in its entirety. While in the first series they measured directly the amount of outflow from the anterior chamber as detected by the air bubble moving away from the eye, in the second series the authors tried to determine the mechanism of reabsorption of fluid from the anterior chamber, raising the pressure of injection gradually from 40 to 120 cm. H_2O. Using intervals of 5 to 10 cm. H_2O they observed that after a period of large variations, liquid flowed into the anterior chamber at a regular rate. At each change of pressure, however, they saw that "a considerable displacement of fluid into the eye took place, due to the distension of the sclera and the expulsion of some blood from the intraocular vascular bed." They found at pressures slightly above the normal (not more than 5 mm. Hg), that the liquid admitted into the eye increased at the rate of from 5 to 10 c.mm. per minute in Experiment 1, that is, about five times more than the normal figure found in the dog, and consider this a proof that the reserve power for the absorption of the aqueous in the anterior chamber is very large. However in Experiment 2 the same increase of pressure of injection gave an increased rate of inflow of only 3 c.mm.

The large variability in the amount of liquid admitted into the eye from Leber's manometer is apparent in the results of Experiments 1 and 2. While in the first at 45 cm. pressure, liquid flowed into the eye from the apparatus at the rate of 10.6 c.mm. per minute, in Experiment 2 at the same pressure, the inflow was only 0.9 c.mm. per minute. At 55 cm. H_2O pressure it was 19.8 and 3.0 c.mm., respectively. The difference persisted until at the end, when 125 cm. of pressure were reached in both, the inflow was 118 c.mm. in Experiment 1 and 22.7 c.mm. per minute in Experiment 2. The size and weight of the dogs had no relation to the inflow, because the animal in Experiment 1 weighed 9.15 kg. while the one in Experiment 2, weighed 14.6 kg.

Any research work made with Leber's manometer is always subject to the same objection mentioned before; that is, the observer assumes *a priori* that the amount injected per minute into the anterior chamber by the apparatus, corresponds to the amount of outflow through the angle and the episcleral veins, which is not really the case. In the living dog the liquid injected into the anterior chamber probably finds a way of escape through the deep ciliioscleral recess that exists between the iris and ciliary body on the inner side and the sclera on the outside, and leaks to the suprachoroidal space and the vitreous.

Increasing gradually the pressure of injection Friedenwald and Pierce obtained a curve of the inflow which consisted of three segments: an initial, sharp rise, a plateau where the inflow remains slightly changed, and another sharp increase, when the pressure of injection is raised from 105 to 125 c.mm. H_2O.

The first two segments of the curve are explained by the mechanism, the authors point out; that is, the first by a considerable increase in the rate of ab-

sorption at a slightly higher than normal, intraocular pressure; the second, or plateau, by a stoppage of the venous circulation when the intraocular pressure equals or is higher than the blood pressure in the veins of the iris, whose walls are compressed until the *vis a tergo* forcibly replenishes and reopens them to absorption, as Duke-Elder had already pointed out. The last rapid ascent of the curve, the authors explain by an escape of fluid through the perivascular lymph spaces around the anterior perforating ciliary veins. But are these lymph channels so wide that they can help to evacuate amounts of fluid as large as 118 c.m.m. per minute? Is it not more probable that the escape of fluid at these great pressures is made through the sinus of the chamber into the ciliary muscle and vitreous, as previously pointed out? There again the fallacy of Leber's method does not permit the conclusion that liquid injected means liquid evacuated from the eye.

In order to prove that their experiments were correct and that there was no outflow from the anterior to the posterior chamber and vitreous, Friedenwald and Pierce ascertained the depth of the anterior chamber by photographing every 30 minutes a beam of light which passed through the optical axis of the eye. No change was observed in the depth of the chamber and the authors conclude that the iris-lens diaphragm remained tight all through the experiment. This is not, however, a complete proof, because as said before, at these great pressures liquid probably found its way backward and distended the eye instead of being absorbed.

The authors also injected a mixture of potassium ferrocyanide and iron ammonium-citrate solution and at the end of two hours the eye was removed and fixed in acid formaldehyde. No deposits of prussian blue were found in the posterior chamber. They do not state, however, whether the ciliosclerał sinus, the muscle, and the suprachoroidal spaces were stained or not.

In the writer's opinion the principal merit of the Friedenwald and Pierce researches is that they have confirmed the existence of a continuous current of aqueous humor into the anterior chamber, and have shown that the flow of this slow current is compatible with the physical laws of diffusion and osmosis, which are claimed by the opposite school to be the sole cause of the production and absorption of the aqueous humor.

350 West Eighty-fifth street.

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THE ARGYLL ROBERTSON PHENOMENON IN MULTIPLE SCLEROSIS

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AND
MICHAEL H. TEITELBAUM, M.D.
BROOKLYN

The authors accept the definition of the Argyll Robertson pupil as formulated by S. A. K. Wilson. Myosis is not considered an integral part of the phenomenon. Until recently, the presence of this type of pupillary reaction was thought to be indicative of syphilis. However, it has been reported in other diseases of the nervous system. In cases of multiple sclerosis it has been mentioned very infrequently. The authors review the literature and report several cases of multiple sclerosis in which the Argyll Robertson phenomenon has been a prominent feature. They conclude that this type of pupillary reaction undoubtedly occurs in multiple sclerosis more often than it has been reported in the literature.

At the outset it is advisable to define the phenomenon to be discussed in this paper, for unless unanimity of definition is reached such expressions as "pseudo-Argyll Robertson pupil" will continue to be used. As Kinnier Wilson[1] points out, some question still exists as to what actually constitutes an Argyll Robertson pupil. The point of contention is whether myosis is or is not to be taken as an integral part of the symptom-complex. Under the title "Four cases of spinal myosis with remarks on the action of the light on the pupil," Argyll Robertson[2] in 1869 published a series of cases of tabes dorsalis in which "the pupils were insensible to light but contracted still further during the act of accommodation for near objects." Myosis was present in all his cases.

A perusal of the standard textbooks on neurology and on ophthalmology indicates that myosis is not considered an essential part of the phenomenon. Yet, Dunn,[3] Adie,[4] McGrath,[5] and others are loath to consider a pupil an Argyll Robertson unless it is also myotic. Present-day opinion, nevertheless, holds myosis to be incidental; so that in the words of Kinnier Wilson the sign may be defined as consisting "in the absence (or obvious diminution) of the direct reflex to light, the consensual being either absent or present, with preservation of the pupillary reaction on convergence accommodation." That this is the soundest view to take is substantiated by the fact that myosis may occur without the dissociated reflex phenomenon of the Argyll Robertson pupil and vice versa. Fuchs[6] estimated that only 25 to 30 percent of the cases of tabes with the Argyll Robertson pupil showed an accompanying myosis, while the phenomenon was also found with a normally wide pupil.

There are those who contend further that the light reflex must be entirely abolished and who do not recognize this phenomenon if any light reaction is preserved. While this may conform to the conception attributed to Argyll Robertson, it is open to modification. Spiller[7] calls attention to the fact that if this were true, it would have to be assumed that the loss of the light reflex is complete from the beginning. This method of reasoning would imply an abrupt onset of the sign and would eliminate all preliminary stages in the production of the Argyll Robertson pupil.

Until fairly recently, it had been a universally accepted fact that the presence of an Argyll Robertson pupil indicated a syphilitic process. Bumke[8] maintained that the Argyll Robertson pupil simply proved the presence of syphilis of the central nervous system. Vinaver[9] was more emphatic in his assertion that a definite case of Argyll Robertson pupil on a nonluetic basis was unknown, and he considered every person presenting this phenomenon to be luetic. Dunn[10] is just as certain that the "complete type of Argyll Robertson pupil cannot be produced—except as the result of syphilitic infection." Holden[11] considers it to be without exception a sign of syphilis of the nervous system,

but he does mention its presence in a reported case of tumor of the third ventricle.

Since 1918 a sufficient number of cases presenting this phenomenon have been reported in nonluetic individuals to cause a modification of the drastic statement that it occurs only in parenchymatous neurosyphilis. Thus, Lutz[12] in 1918, stated that "undoubtedly we find typical Argyll Robertson phenomenon very exceptionally in certain nonsyphilitic diseases such as sclerosis multiplex, syringo-myelia, etc. But these are all rare exceptions." While it is Ingvar's[13] opinion that the presence of this sign suggests at once a syphilitic etiology, he admits that the reflex pupillary rigidity may occur in disseminated sclerosis. "But we know that in this disease this symptom is not only atypical but is a great rarity even in the most advanced stages."

Further, the finding of the Argyll Robertson phenomenon in epidemic encephalitis and in other diseases caused a change of opinion regarding the specificity of lues as the sole etiological factor. At present we are safe in assuming that neurologists do not consider the Argyll Robertson pupil alone diagnostic of neurosyphilis. A perusal of the literature reveals conflicting reports as to its presence in multiple sclerosis. Oppenheim[14] finds that fixed pupils may rarely occur in this disease but says nothing of the Argyll Robertson phenomenon. The Oxford System of Medicine[15] fails to mention the possibility of such a pupil in multiple sclerosis, while Taylor[16] in Nelson's Loose Leaf Medicine thinks that an "Argyll Robertson pupil presumedly does not occur" (in multiple sclerosis). Lagrange and Marquézy,[17] observing 52 cases, and Velter[18] reporting a similar number, are of the opinion that this type of pupillary reaction does not occur in multiple sclerosis. In a statistical study of 206 cases of multiple sclerosis, Berger[19] concluded that differences in size of the pupils occur very frequently, though usually to a limited degree. However, he noted variations in 30 cases. On the whole, the reaction to light was normal; nevertheless in 12 instances it was sluggish, and in 2 of these the reaction to accommodation was also sluggish. In 2 cases the pupils were fixed to light. No true Argyll Robertson pupils were noted.

At the meeting of the Association for Research for Nervous and Mental diseases in 1921, Sachs and Friedman,[20] in listing the signs and symptoms of multiple sclerosis, stated that "pupils [were] generally active with occasional hippus. Pupillary disturbances noted rarely." In replying to a question as to whether an Argyll Robertson pupil could occur in multiple sclerosis, Sachs stated that "As far as the Argyll Robertson pupil is concerned, my feeling is that it occurs so rarely in any other condition except in one of the luetic degeneration diseases of the brain or cord, that I am willing to say that in multiple sclerosis it never occurs; in fact if I saw a case I otherwise considered multiple sclerosis and that person had an Argyll Robertson pupil, I should be greatly inclined, on account of that, to consider it out of the category of disseminated sclerosis." Wechsler[21] concludes that "It is debatable whether a true Argyll Robertson pupil is ever encountered in uncomplicated multiple sclerosis. . . . Indeed it may be said that the presence of disturbed pupillary reactions is fairly good evidence against the disease" (multiple sclerosis).

However, a careful search of the literature reveals that cases of multiple sclerosis have been reported in which the Argyll Robertson phenomenon was a significant feature. As far as possible the original source of each reported case was read in order to ascertain whether it was one of undoubted multiple sclerosis and whether the pupillary disturbances withstood the test of the definition as formulated by Kinnier Wilson. In 1890, Uhthoff[22] mentioned Parinaud as having been the first to study thoroughly the pupillary disturbances in multiple sclerosis. Uhthoff himself observed 100 cases, of which 11 percent showed abnormal pupillary light reactions. A very slight yet definite pathologically decreased reaction to light was seen in 5 percent. The various pupillary disturbances listed by him follow:

1. Pupils fixed to light with myosis: 1 case.
2. Myosis with preserved but limited reaction to light and to accommodation: 4 cases.
3. Light reaction very markedly diminished without myosis: 1 case.
4. Marked difference in the width of the pupils: 3 cases, 2 of which showed diminution of the light reaction.
5. Reaction to convergence diminished, with relatively good light reaction: 2 cases.

While Uhthoff does not mention the reaction to accommodation in the first group, one feels justified in assuming it was unimpaired, for in the remaining groups he calls attention to it when the reaction is abnormal.

In 1901 Probst[23] reported a case of multiple sclerosis which he had observed for 19 years. Autopsy confirmed the diagnosis. At the time of admission to the hospital the pupils were equal, round, and reacted well to light. Differences in the size of the pupils soon appeared, so that the left pupil was twice as large as the right one. In the meantime the light reaction became extremely sluggish; the reaction to accommodation was sluggish but to a lesser degree. In the seventeenth year of the disease the difference in the size of the pupils disappeared, but the reaction to light remained sluggish. Subsequently the pupils became myotic and light fast. The sluggish reaction to accommodation would seem to make them incompletely fixed pupils rather than Argyll Robertson. Yet later writers (Wilson, Rad) consider this a case of multiple sclerosis presenting the Argyll Robertson phenomenon.

In a case reported by Pini[24] in 1903, the pupillary findings were as follows: inequality in size; light reaction in the right eye abnormal, and almost completely lost in the left eye. At a later examination the light reaction in the left eye was present but sluggish. Even though there were no serological determinations, Pini feels certain that his case was one of multiple sclerosis. Unfortunately he neglects to give us an inkling of the reaction to accommodation.

Marburg[25] in 1906 presented a case of "So-called acute multiple sclerosis (encephalomyelitis periaxialis edematous)" in which the pupils reacted very sluggishly to light and the reaction to accommodation was questionable. Similar eye findings were noted in a case of Henschen's[26] in which an acute multiple sclerosis with neuritis developed in a child after an attack of diphtheria.

Stadelmann and Lewandowsky[27] reported a case in which the diagnosis oscillated between acute multiple sclerosis and disseminated myelitis, the authors favoring the former diagnosis. The pupils were dilated; the left more than the right. The former did not react at all to light, the right reacting slightly.

Writing "On the loss of the light reflex in multiple sclerosis," Rad[28] in 1911 reported 2 cases. In the first the pupils were in midposition and did not react to light, the reaction being tested in a darkened room. The reaction to convergence was good. In the second case the pupils were somewhat narrow and absolutely fixed to light. The reaction to convergence was good. Serology was completely negative in both instances. There can be no doubt that both were clinically cases of multiple sclerosis and that the pupils gave true Argyll Robertson reactions. Rad mentions 2 cases reported by Liwschütz[29] in which the reaction of the pupils to light was absent. Since an optic atrophy was present, however, in one of these cases, Rad thinks that the loss of the light reflex might have been due to it. He also doubts the correctness of the diagnosis in this case. He calls attention to the monograph of Edward Miller[30] who reviewed the 364 observations of multiple sclerosis collected by Von Frankl-Hochwardt,[31] Uhthoff, and Probst and found the Argyll Robertson phenomenon recorded in only 4 instances. In his own rich material, Miller saw none, though one had a sluggish reaction to light.

Anton and Wohlwill[32] found some difficulty in determining whether to call their cases multiple sclerosis or encephalomyelitis. In the first one the left pupil was wider than the right and reacted somewhat sluggishly to light but well to accommodation. Later the reaction to light was normal. Subsequently the pupils did not react at all to light.

The left somewhat later reacted distinctly to light. Sometime before death the right one reacted slightly to light and well to accommodation. In the second case the left pupil reacted sluggishly to light at the beginning and well to accommodation. Later the right was found to be narrower than the left, and to react almost imperceptibly to light and slightly to accommodation. The left pupil reacted well to light.

Kinnier Wilson[1] reported a case in 1921 of multiple sclerosis which he thought presented an Argyll Robertson pupil. "Both pupils were rather dilated (diameter 5 mm.) and completely inactive to strong light but they reacted quickly on convergence. Optic discs normal." Reporting a case of encephalomyelitis which he thought might have been multiple sclerosis, Deusch[33] noted at first the following disturbances: absolute rigidity of the pupils to light; fundi pale. A few weeks later the light reaction was still absent. However, this was followed by a return of the reaction to accommodation. Bramwell[34] writing in the "Osler system of medicine," states that "Inequality of the pupils is not uncommon in multiple sclerosis. The light is rarely lost or impaired unless in the presence of a scotoma. The writer has met with one instance of an Argyll Robertson pupil in multiple sclerosis."

In 1930 Guillian, Cournand and Rouques[35] reported a case of acute disseminated sclerosis (encephalomyelitis) with an Argyll Robertson pupil of transient occurrence. At first the pupils were unequal; then they became equal. "The Argyll Robertson sign was noticeable on the left side; it progressed and at the same time the light reflex on the right side became almost zero. In the third phase, the light reflex in the right eye was almost normal again, although that of the left eye still remained weak. The reaction to accommodation was constantly normal. Blood Wassermann and spinal fluid were negative."

Cases. To this series we wish to add 3 cases of multiple sclerosis presenting the Argyll Robertson phenomenon which have come under our observation in the past 2 years:

Case 1. M. L., male, aged 40 years, was admitted to the hospital because of polyuria, loss of sphincter control, numbness of the hands and feet, weakness of the legs, and dimness of vision for the past 10 years. Loss of sexual power had occurred 4 years ago. For 2 years he had found himself unable to walk. The present illness began about 10 years ago with weakness in the legs which extended upward to involve the hips and thighs, so that he was unable to walk without the aid of a cane. Later he would fall if not supported; and for the past 2 years he had not been able to walk at all. The numbness of the hands and feet began soon after the onset of the illness. The past history was irrelevant. Physical examination revealed the following: speech was somewhat mumbled and definitely scanning. The pupils were contracted, equal, did not react to light at all but did react to accommodation. There was a horizontal nystagmus on looking to both the right and left. Fundus examination showed a temporal-half pallor of the right disc and a complete pallor of the left disc. A central scotoma was present in the left eye. There was no cranial-nerve involvement. An intention tremor was present in both hands, but more marked in the left. The abdominal reflexes were not elicited. The knee-jerks were over-active, and a bilateral Babinski reflex and inexhaustible ankle clonus were found. Vibratory sensation was absent in the lower extremities, of which there was also an apparent ataxia. Motor power was diminished in all limbs. The laboratory findings were as follows: urine showed an occasional hyaline cast; blood and spinal fluid Wassermanns were negative; colloidal gold curve showed no reduction; globulin and sugar were normal in the spinal fluid, with no cells. The blood findings were within normal limits. Diagnosis: multiple sclerosis. A similar diagnosis had been made in another hospital.

Case 2. J. M., male, aged 55 years, was admitted to the hospital because of anuria for 24 hours and difficulty in urinating for 6 years. For the past 4 years he had experienced difficulty in the starting and in the cessation of the urinary stream. He had had nocturia and some pain on urination. Just before admission to the hospital he had found himself unable to void and had to be catheterized. In addition to the urinary complaints he remarked that as long as 15 years ago he had noticed weakness of the right lower extremity, followed soon afterwards by a weakness of the right arm. At the time of admission there was still a weakness of the right leg, so that he had to use a cane with which to walk. The weakness of the right arm had cleared up. The family and marital history were unimportant. Physical examination showed the following: right pupil slightly irregular and larger than the left. Neither reacted to light but did to accommodation. The eye grounds were normal. There was a horizontal nystagmus. The cranial nerves were not involved. Bilateral Babinski reflex was present, more marked on the right, and an

inexhaustible ankle clonus. All deep reflexes in the lower extremities were accentuated. Speech was hesitant but not definitely scanning. The abdominal reflexes were not elicited. A slight intention tremor was present in the right upper extremity. Sensation in all forms was intact. Urological consultation revealed a prostate within normal limits for his age, and the opinion was expressed that the retention was most likely due to neurological pathology. Cystoscopic examination showed a low-grade cystitis. The blood chemistry was within normal limits. Urinalysis showed an occasional r.b.c. but no casts or w.b.c. Blood and spinal-fluid Wassermanns were negative. Diagnosis: multiple sclerosis.

Case 3. T. B., female, aged 50 years, was admitted to the hospital because of generalized stiffness of the body and inability to move the legs. The present illness dated back at least 28 years, and was said to have begun after a fall. She awoke the morning following the fall and found a marked weakness of both legs together with a loss of power in the right arm. Since the onset of her illness there had been periods of very slight improvement; but for the past few years she had become progressively worse, so that at present she was a wheel-chair patient. A tremor of the left arm developed soon after the onset of the illness. She had had 2 residences in other hospitals, in one of which a high cervical-cord neoplasm was suspected. Operation, however, disclosed no pathology of the cord, macroscopically. Physical examination at the time of admission to the hospital revealed the following: The patient was an obese, adult female, whose speech was not scanning. The pupils were contracted almost to pin-point size, equal and regular; they reacted to accommodation, but very slightly, if at all, to light. The fundi were normal. No nystagmus nor cranial-nerve involvement was observed. There was a weakness and spasticity of the right arm and an almost complete loss of power with marked spasticity of both legs. Biceps and knee-jerk reflexes were very active. Bilateral Hoffmann's sign and bilateral Babinski and ankle clonus reflexes were elicited. Ataxia was indicated on the finger-to-nose test. There were no sensory levels, though there was a right-sided hypalgesia. The sensation of vibration was diminished in the lower extremities, especially on the right side. The gait was spastic. Abdominal reflexes were not elicited. The Wassermann reaction was negative in the blood and in the spinal fluid. Diagnosis: multiple sclerosis.

Discussion

The cases presented by us have been under our observation for a long time. The disease process has progressed sufficiently to make the diagnosis of multiple sclerosis a clinical certainty. The patient's reaction to light in each case was tested in a darkened room. A strong electric light was flashed into the eye, the patient accommodating for distance. We think that for the sake of accuracy the method of examination should be described when the reaction to light is reported as abolished or obviously diminished. From the presentation of the foregoing cases it is apparent that a characteristic Argyll Robertson pupil is occasionally found in diffuse toxo-infectious states other than syphilis. It would indeed be surprising if this were not so. Kinnier Wilson expressed this thought when he said "A diffuse morbid process will sometimes light on the same area and disturb the same morbid mechanisms as are implicated in the Argyll Robertson pupil of syphilis."

However, while we are in accordance with this view it is not entirely subscribed to by some authors, particularly Ingvar. In his admirable paper on the subject he agrees with Behr[36] that only a lesion immediately rostral to the anterior quadrigeminal bodies is capable of producing the Argyll Robertson phenomenon. From an evolutionary basis he argues that one has to look for the pupillary pathways on the surfaces of the diencephalon. The pathways run in immediate contact with the meninges on the very surface of the basal brain region. Consequently, Ingvar feels that only such diseases as cause marginal destruction within the basal subarachnoid spaces of the brain can produce the Argyll Robertson pupil. Syphilis causes such marginal destruction. Multiple sclerosis, on the other hand, even in the advanced stages of the disease, shows a tendency to spare the peripheral apparatus, although there are numerous inflammatory foci disseminated over the rest of the nervous system.

Dunn also ascribes to syphilis the prime role in the production of the Argyll Robertson pupil. He locates the lesion in the ciliary ganglion. Ferrier[37] is in accord with him; Ingvar, Lutz, Karplus and Kreidl[38], Behr[36], Barkan and Mehrents[39], Adler[40] and others have added much to our knowledge of the anatomy of the pupillary pathways and the pathogenesis involved in the production of the Argyll Robertson pupil.

However, in this paper we are not primarily concerned with this phase of the problem.

Our cases would seem to bear out the contention of Kinnier Wilson that this type of pupillary reaction does occur in undoubted cases of multiple sclerosis. We believe, moreover, that it occurs in many more cases of multiple sclerosis than have been reported as presenting this phenomenon. Early and incomplete states of this type of pupillary reaction have frequently been discarded as "pseudo-Argyll Robertson" and probably no attempts have been made to reexamine the pupils in the late stages of the disease to ascertain whether a change in the light reaction has taken place as the disease progressed. The case reported by Probst illustrates this. In the 19 years that the patient was under his observation progressive changes took place in the pupillary reactions, until the pupils finally became fixed to light and were miotic. A patient seen by Nonne and Schreiber[41] showed a similar metamorphosis, passing from a normal reaction to the Argyll Robertson state while under their observation.

In reviewing the original report of cases of multiple sclerosis said to present this phenomenon, one is lead to the conclusion that many of these do not fall into the category of what is commonly considered multiple sclerosis. We have in mind the chronic, progressive type of disease, episodic in nature with gradual progession and temporary remissions. Stern[42] feels much the same way when he writes that of the few reputed cases of multiple sclerosis presenting the Argyll Robertson pupil, only the one described by Nonne and Schreiber exhibited a persistent and positive Argyll Robertson pupil. He apparently has overlooked the cases of Rad whose report leaves no doubt as to their authenticity. We were not able to locate the case of Nonne and Schreiber mentioned by Stern.

Since the exact status of the so-called acute multiple sclerosis is still in doubt, one hesitates to include in this series the reports of Marburg, Henschen, Stadelmann and Lewandowsky. Further, since the relationship between multiple sclerosis and encephalomyelitis has not been satisfactorily determined, one cannot accept without reservation the cases reported by Anton and Wohlwill, and Liwschütz. Pini leaves something to be desired in his description of the pupillary findings. The absence of a definite statement regarding the reaction to accommodation makes the pupil a doubtful Argyll Robertson type although the other writers consider that such a pupil was present in these cases.

Conclusions

1. Kinnier Wilson's definition of an Argyll Robertson pupil is accepted as a working basis for this paper.

2. The Argyll Robertson phenomenon is not an indisputable sign of syphilis when it occurs alone.

3. It is found in undoubted cases of multiple sclerosis (Rad, Wilson).

4. This type of pupillary reaction is probably present in more cases of multiple sclerosis than have been reported in the literature.

5. We believe that the three cases we have presented are clinically multiple sclerosis.

6. All exhibit pupils which come under the definition of an Argyll Robertson as formulated by Kinnier Wilson.

7. This phenomenon is still a rare finding in cases of multiple sclerosis.

626 Linden Blvd.

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SIMULATION AND OCULAR HYSTERIA

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The essential difference between the simulator and the hysteriac is his mental state: in both, the subjective and objective pictures are incongruous, but in addition the simulator tries to mislead, is apprehensive under examination, nonco-operative, and contradictory; the hysteriac suffers from psychic abnormality but is desirous of regaining his health and, hence, is co-operative. There is a third condition, that of the hysterical simulator, which is much more difficult to diagnose and uncertain as to cure. Presented at the First Latin-American Ophthalmological Congress.

Traumatic ocular injuries give rise to two types of patients: those with a visual deficiency and ocular changes, and those with a visual deficiency without ocular changes, in whom the positive subjective examination is accompanied by a negative objective examination. In this paper only the second clinical type will be treated. It offers two diagnostic possibilities: simulation and hysteria. The differential diagnosis is sometimes very difficult, and it sometimes happens that a judge is compelled to give his decision even though the medico-legal reports are contradictory.

To begin with, it is well to bear in mind that very frequently the ocular examination does not give the key to the solution; in fact, simulation and hysteria are identical in symptomatology: a local functional deficit together with anatomical integrity. In practice, as a rule, the little, familiar signs of narrowness of the visual field, discromatopsy, and so forth, are not those that will resolve the problem. It is therefore necessary to make a general examination, or better still, a psychological one.

What, then, is the essential difference between the simulator and the hysteriac? His mental state. The hysteriac suffers from psychic abnormality, from neurosis; he is abnormal. The simulator is a healthy person who presents a moral abnormality. He tries to mislead; he is a fraud. There is something secret about him that separates him completely as a clinical entity from the other. This secret the physician must discover, for as soon as he finds it the diagnosis is clear. What is it? Just what the patient himself thinks of his illness.

The hysteriac thinks that he is really ill and ardently desires to be well. The simulator knows that he is well and will not admit it, for the admission would cause him pecuniary loss. These two different mental states show diverse attitudes between the simulator and the hysteriac in the presence of the physician. To the simulator the doctor is an enemy and he treats him as such. The doctor interposes between him and indemnification. To the hysteriac the doctor may be a savior, the only one who can give him back his health, and he comports himself respectfully and humbly before him.

The simulator presents one of two clinical types: one is that characterized by his wearing apparel, the other is not externally characterized. We will describe them in order.

The type characterized by his wearing apparel: To make his invalid condition more convincing the simulator adorns it with external manifestations. He tries to give the impression of blindness. He dresses like the artist in the theater who represents a sick man, using large black goggles, a slouch hat over his eyes, and so on. He is a comedian.

In the clinic of the Salvador Hospital we are making a gallery of simulators (fig. 1). Some of these patients had deceived several doctors and presented convincing certificates in their favor. The diagnosis of this clinical type comes to mind the moment the patient steps into the consulting room.

The type not externally characterized: This patient simulates less and

Fig. 1 (Charlin). Simulating amaurosis.

yet he simulates better, but he betrays himself in cross-examination. We have said that for the simulator the doctor is an enemy, and as such he treats him. The patient has an impatient, indocile, hostile attitude; he answers roughly, or in monosyllables, or evasively, the questions put to him. He feels in danger at every question; each test is a threat that might uncover his deceit. He undergoes examination unwillingly and tries to shorten it, arguing that the ophthalmoscope's light hurts his sight. This is never seen in the hysteriac, who has a certain pleasure in being examined.

The same two types are found in the hysteriac, the type externally characterized being the grand ocular hysteriac; the other has no external characteristics.

The grand ocular hysteriac has an unusual attitude. He does not move, but remains for a long time in certain positions, whether sitting or standing, ecstatic (*figé*, fixed), staring at the floor as if he were absorbed in a great preoccupation. He suffers from a kind of catatonia. Besides this, there is a certain rigidity; the head is slightly bent, and he maintains a continual muteness. The face is expressionless. This immobility, rigidity, and lack of expression might make one think of encephalitis lethargica, or of Parkinson's disease (fig. 2).

Fig. 2 (Charlin). An hysteriac after ocular trauma.

The type without external characteristics is difficult of diagnosis, for nothing outwardly reveals the psychic abnormality, the neurosis. Frequently there are slight signs of ocular hysteria, such as blinking, the permanent contraction of the frontal muscles, and

a slight ptosis (fig. 3). But even when there is amaurosis or amblyopia in one or both eyes, diplopia in one or both eyes, contractions, ocular paralysis, ptosis, and so forth, there is something peculiar to ocular hysteria; namely, its absurd symptomatology. The pathologic alteration presented by the patient does not endure serious criticism. The hysterical symptom cannot be sustained, for it is not supported either anatomically or physiologically. As soon as the symptom is studied it falls to the ground and presents an absurd clinical evaluation.

I remember, among others, a patient who complained of blindness in the left eye, which made him assume an unnatural position of the head in order to see only with his right eye—a vicious position of the paralyzed rectus. Now, such a position of the head is for the purpose of overcoming diplopia, which does not exist in a case of unilateral amaurosis. Besides this, the patient, in order to see better, widely opened his eyes, contracting the frontal muscles—another absurd attitude, for to improve sight we naturally contract the lids so as to reduce the orifice in the ocular diaphragm. Sometimes this absurd clinical picture is not so noticeable and can be brought into evidence only after a careful anamnesis; then the clinical

Fig. 3 (Charlin). A case of ocular hysteria with bilateral blepharospasm.

Fig. 4 (Charlin). Bilateral hysterical amaurosis. The patient is in an attitude of complete indifference.

characteristics, the relations of the disease, as described by the patient, are found to be incongruent, fantastic, unreal.

I have had cases in which I have diagnosed hysteria just by questioning the patient. I remember a young society girl in Santiago, the despair of the physicians who had seen her, who had kept her family in constant alarm. For a long time she had had high intracranial pressure, tenacious headaches, daily vomiting for six months, and visual disturbances. A colleague sent her to me to be examined for papillary edema. Knowing her past history, I looked at the patient and saw a rather stout young lady, rosy and smiling. Laughingly she spoke of her headaches and the daily vomiting. The patent contradiction between the anamnesis and her general condition, the absurd clinical picture, could not be real, for we never see patients in whom headaches and vomiting have lasted for months whose general condition is not compromised, and this made me think of hysteria. The patient had no papillary edema; she had a sensitive-sensorial anesthesia.

Bilateral hysterical amaurosis, reputed by some writers to be very difficult of diagnosis at times, is often uncovered by this characteristic, absurd clinical picture. The hysterically blind patient assumes the attitude of a deaf man who is not deaf. If you speak to him, he answers the questions, but never looks at his questioner. He thinks he is acting wonderfully as a blind person (figs. 4 and 5). The truly blind man turns his face toward the person to whom he speaks or who is speaking to him, although he cannot see him. This attitude, this clinical incongruity, is enough to diagnose hysterical blindness. In addition, this peculiar position will equally well betray unilateral hysteric amaurosis, for when the good eye has been bandaged and the patient is addressed, he will assume an attitude of deafness.

Fig. 5 (Charlin). The patient with organic blindness turns to answer his interlocutor; the hysteriac assumes an attitude of deafness.

Before finishing this description, the intermediate type should be mentioned, one very difficult of diagnosis, and very uncertain as to cure; namely, the hysterical simulator. This is the patient who after an accident, for example, remains with an imaginary visual deficiency. He believes in his visual invalidism and does not see; in this respect he is hysterical. But he knows, he has been told, that this invalidism might bring him economic benefit and for that reason he is opposed to cure, to suggestion; in this respect he is a simulator. His opposition, however, is really unconscious, so that again he is an hysteriac.

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THE BRILLIANCE OF AN OBJECT SEEN BINOCULARLY

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On the hypothesis that pathways from each pair of corresponding points of the two retinae converge upon a common pathway in the brain and that physiological summation takes place, the authors explain their experimental finding that binocular vision gives an increase of brilliance over monocular vision. From the Department of Ophthalmology, Oscar Johnson Institute, Washington University, St. Louis.

Introduction

Although some investigators have pointed out that an object seen binocularly appears to be more brilliant than when seen with one eye, some doubt is still current whether this is true. In this paper further evidence is presented that binocular vision gives an increase in brilliance, and it is shown that this fact can be accounted for on the hypothesis that pathways from each pair of corresponding points of the two retinae converge upon a common pathway in the brain and that physiological summation takes place.

It is often argued that this hypothesis is contradicted by Fechner's paradox, which consists in this, that if a given area of one of the retinae, say, the right retina, is stimulated with a certain intensity, and the corresponding area of the left retina is more feebly stimulated, this feeble stimulation of the left retina adds nothing to the net brilliance, but diminishes it. Now if summation were the only factor involved, the feeble stimulation of the left retina should add something, however little, to the net brilliance; but summation is not the only factor. It is aimed to show in this paper that when the role played by border contrast is taken into account, Fechner's paradoxical effect is compatible with the hypothesis stated above.

Graham[1] recently showed that the absolute threshold (the minimal stimulus that will produce a sensation) is no lower for two eyes than for one. This finding is compatible with the hypothesis of summation at a common cerebral pathway, as is shown by the following consideration: First of all, two thresholds must be taken into account; namely, (1) the minimum amount of light to produce a response in either of the two converging pathways, and (2) the minimum frequency of impulses arriving at the common cerebral pathway to produce a discharge. In the case of either one of the two converging pathways there must be a certain minimum frequency of impulses below which it will not respond at all to stimulation of the retina by light. And if this frequency is sufficient to evoke a discharge in the common cerebral pathway, there is no occasion for adding a stream of impulses via the other converging pathway. In other words, a possible interpretation of Graham's finding is that the absolute threshold depends entirely upon the threshold at the retina, and can therefore throw no light upon the problem of the threshold at the common cerebral pathway.

I. Apparatus and procedure

By means of a head rest the eyes of the observer were fixed in the position indicated in figure 1. The two eyes fixated the center of a white card, K, attached to the front of a black box, which was 12″ away. In the card were two rectangular holes, A and B (¼″ × ½″ and ¼″ apart), one above the other, which were illuminated from behind as illustrated, and appeared not as holes, but as surfaces in the plane of the card.

V, an opal glass screen, was the light source for rectangle B for both eyes. The light from V was reflected by a mirror in the lower half of the box through rectangle B to both eyes. B was, therefore, equally bright in the direction of either eye. The brightness of B could be varied gradually by the observer

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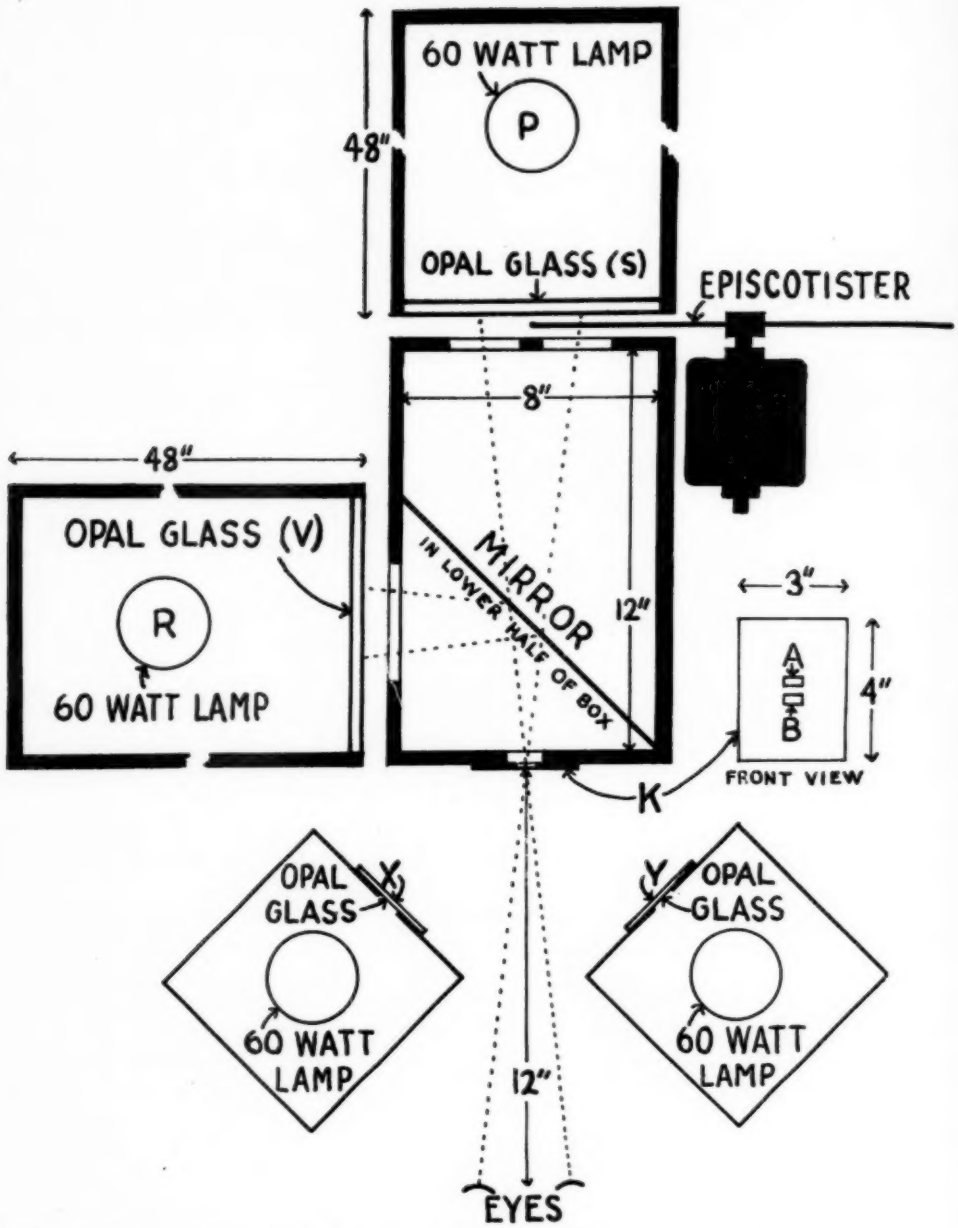

Fig. 1 (Fry and Bartley). Diagram of apparatus. The white card, K, is illuminated by light sources, X and Y. The light from opal glass, V, is reflected by the mirror in the lower half of the box through hole, B, to the eyes of the observer; and the light from opal glass, S, passes through hole, A. The episcotister reduces the brightness of A as seen by the left eye. The observer's procedure consists in adjusting the position of lamp, P, so that A and B appear equally brilliant.

while observing. S, a similar screen, whose brightness was set at various levels by varying the position of lamp P, was the light source for rectangle A as seen by both eyes. The brightness of rectangle A as seen from the direction of the left eye could be varied by an episcotister and in this way could be made to differ from the brightness as seen from the direction of the right eye. For the sake of convenience the symbols, AR and AL, will be used to refer to rectangle A as seen by the right eye and the left eye respectively.

Our procedure consisted in setting the brightnesses of AL and AR at given levels and in having the observer adjust the brightness of B until A and B appeared to be equally brilliant. Ten of these adjustments were made for every combination of AL and AR, and the average brightness for the ten adjustments was regarded as a suitable measurement.

The brightness of the ground surrounding A and B could be set at various levels by regulating the two light-sources, X and Y.

Artificial pupils were not used.

II. Binocular vs. monocular brilliance

Various investigators, including Aubert, Valerius, and McDougall, have pointed out that an object seen binocularly appears to be more brilliant than when seen monocularly. Sherrington[2], however, found that binocular vision adds nothing to brilliance, but since his apparatus was not adequately adapted for an investigation of this problem his findings may be disregarded. With a much more suitable apparatus DeSilva and Bartley[3] have demonstrated that in order for an object seen monocularly to appear just as brilliant as an object seen binocularly, it must be 1.27 to 1.44 times as bright, depending upon the experimental situation. Further data on this point are furnished by the following experiment:

Experiment 1. A piece of black cardboard was placed in front of the left half of screen S (fig. 1) so that A appeared black to the right eye. The ground surrounding A and B was made black also. The brightness of AL was set at various levels, and at each level the observer determined the brightness of B at which A and B appeared to be equally brilliant. In every case it was necessary to reduce the brightness of B much below the brightness of AL. The results are plotted in graph I in figure 2.

Two of Fry's curves are presented. In the case of the dots the measurements were made in the order from right to left, and vice versa in the case of the crosses. The difference between the two curves reveals the influence of adaptation which undoubtedly comes into an experiment of this kind. A, being much brighter than B, fatigues the retina more quickly, and toward the end of a series of observations A is less able to compete with B in brilliance than when the two are applied to a fresh retina. In the case of Bartley's curve the measurements were made in the order from right to left.

The fact that binocular vision gives an increase in brilliance can be satisfactorily accounted for on the hypothesis that pathways from corresponding points in the two retinae converge upon a common pathway in the brain and that summation takes place.

Sherrington, who found that binocular vision gives no increase in brilliance, argued from this that the pathways from corresponding points of the two retinae are independent of each other up to and including those points where consciousness is adjunct, and that single vision with two eyes depends upon the psychical integration of independently elaborated monocular images. Even if it is admitted that binocular vision adds to brilliance, this fact might be accounted for on the hypothesis of physiological independence by saying that "psychical summation" takes place. But once the phenomenal fact of summation is admitted, the hypothesis of physiological independence loses one of its strongest supports.

III. The relation of border-contrast to the brilliance of an object seen binocularly

The physiological process subserving border contrast is little undestood[4], but it is reasonable to suppose that it involves interaction between neighboring retinocortical pathways. Such interaction might take place at the retina, at the basal ganglia, or at the cortex. Now if the border contrast process has its seat in the cortex where pathways from

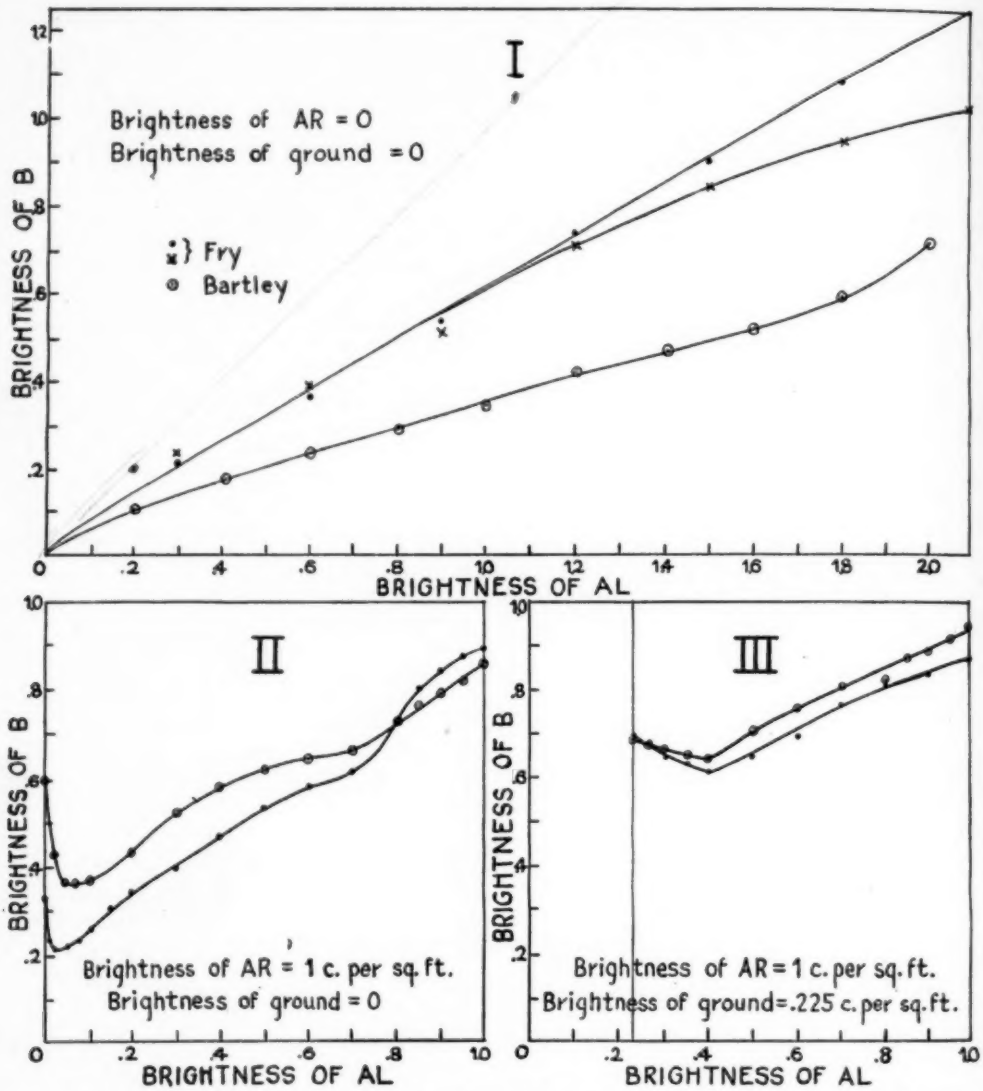
I
Brightness of AR = 0
Brightness of ground = 0
Fry
Bartley
BRIGHTNESS OF B
BRIGHTNESS OF AL
II
Brightness of AR = 1 c. per sq. ft.
Brightness of ground = 0
III
Brightness of AR = 1 c. per sq. ft.
Brightness of ground = .225 c. per sq. ft.

Fig. 2 (Fry and Bartley). Graph I shows how the brightness of an object, B, seen binocularly compares with the brightness of an object, A, seen monocularly when both appear to be equally brilliant. Graphs II and III show the effect of varying the brightness of the ground upon Fechner's paradox.

the two eyes converge, one might expect this process to affect the manner in which the two converging streams of impulses are integrated. That border contrast affects the brilliance of an object seen binocularly can be shown clearly in the case of Fechner's paradox. The demonstration of this involves the two following experiments:

Experiment 2. The brightness of the ground was kept constant at 0; the brightness of AR was kept constant at 1 c. per sq. ft.; and the brightness of AL was set at various levels ranging from 0 to 1 c. per sq. ft. For each combination of AL and AR the observer determined the brightness of B at which B and A appeared to be equally brilliant. The results for two observers are plotted in graph II in figure 2.

Beginning at 0, as AL is gradually increased in brightness, up to a certain point the brilliance of A decreases, but beyond that point it begins to increase and finally reaches a brilliance much greater than the original.

If summation were the only factor involved in the integration in the brain of a sinistrocular with a dextrocular process, one would be led to suppose that the brilliance of A would gradually increase from the very beginning without first undergoing the paradoxical decrease.

Experiment 3. The brightness of the ground was kept constant at .225 c. per sq. ft.; the brightness of AR was kept constant at 1 c. per sq. ft.; and the brightness of AL was set at various levels ranging from .225 to 1 c. per sq. ft. For each combination of AL and AR the observer determined the brightness of B at which A and B appeared to be equally brilliant. The results for two observers are plotted in graph III in figure 2.

If AL is lowered below the brightness of the ground, rivalry between AL and AR takes place. Hence the brightness of AL which is equal to the brightness of the ground must be taken as the starting point. As AL is gradually raised above the brightness of the ground, the brilliance of A decreases at first but then begins to increase and finally reaches a value much greater than the original.

A comparison of experiments 2 and 3 shows that the paradoxical decrease in brilliance depends not upon the brightness of AL taken absolutely, but upon the difference between its brightness and that of the ground; that is, upon the existence of a border between AL and its ground.

Therefore it seems in order to postulate that the physiological process subserving border contrast between AL and its ground in some way depresses the dextrocular process initiated by AR, and it might be supposed that the dextrocular process depresses the sinistrocular process in turn.

Let us return now to experiment 2 and present a complete theoretical analysis of what takes place. In the diagram, figure 3, curve C represents the dextrocular activity initiated by AR; it remains constant because the brightness of AR remains constant. Curve D represents the sinistrocular activity initiated by AL; it is assumed that it increases gradually as the brightness of AL increases. Curve E represents the amount by which the dextrocular process depresses the sinistrocular activity when the two monocular processes meet in the brain. Curve F represents the amount by which the sinistrocular process depresses the dextrocular activity. If these four curves are added together algebraically, the resulting curve, G, represents the net amount of activity resulting from summation of the two monocular activities, their depressive effects upon each other being taken into account. This curve corresponds closely with the curves in graph II, which represent net brilliances resulting from the various combinations of AL and AR.

This analysis shows how Fechner's paradox arises. When the brightness of AL is .05 c. per sq. ft. Fechner's paradoxical effect reaches its maximum. Here the dextrocular activity is appreciably depressed by the sinistrocular process, and this depression is not compensated by the small amount which the sinistrocular activity adds to the dextrocular by summation.

When the brightness of AL and the brightness of AR are both 1 c. per sq. ft., the two monocular processes, in combining in the brain, exert depressive effects upon each other, but these effects are more than compensated by summation, the net result being that AR plus AL produces a greater brilliance than AR by itself.

The following experiment was performed to ascertain what happens when

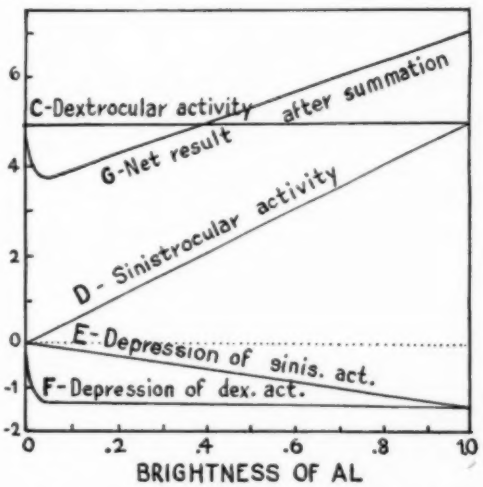

Fig. 3 (Fry and Bartley). An analysis of the factors involved in Fechner's paradox. The values of the ordinates are only estimated relative values, + values representing amounts of activity, and — values amounts of depression of activity.

a dark object on a white ground is presented to one eye and a uniform white ground is presented to the corresponding parts of the other:

Experiment 4. The brightness of AR and the brightness of the ground were both kept constant at 1.23 c. per sq. ft., and the brightness of AL was set at various levels ranging from 0 to 1.23 c. per sq. ft. For the various brightnesses of AL the observer determined the brightness of B at which B and A appeared to be equally brilliant. The results are plotted in graph IV in figure 4. As the brightness of AL increases the brightness of B increases almost proportionally.

The object of the following experiment was to find what happens when a light-gray object on a white ground is presented to one eye, and a dark-gray object on a white ground is presented to the corresponding parts of the other:

Experiment 5. The brightness of AR was set at 6.95 c. per sq. ft. and the brightness of the ground at 12.3 c. per sq. ft. The brightness of AL was set at various levels ranging from 0 to 12.3 c. per sq. ft. For each combination of AR and AL the observer determined the brightness of B at which B and A appeared to be equally brilliant. The results

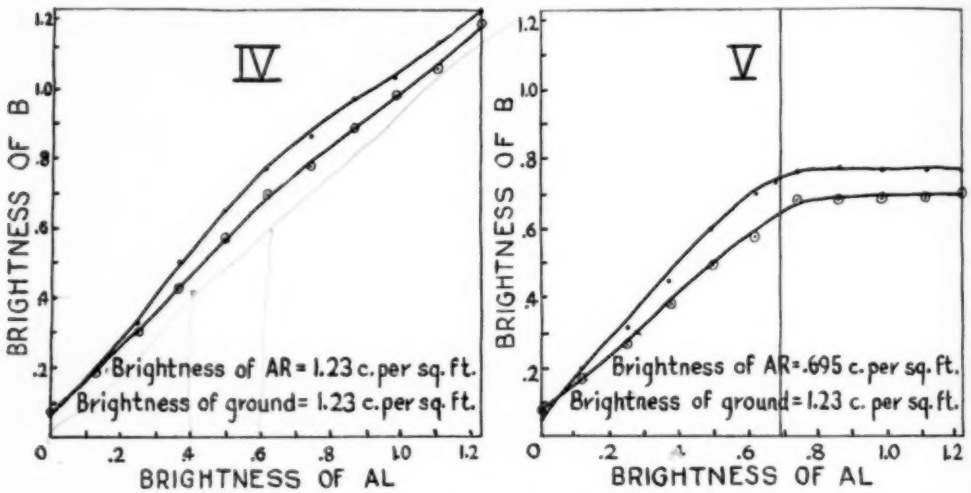
IV
BRIGHTNESS OF B
BRIGHTNESS OF AL
Brightness of AR = 1.23 c. per sq. ft.
Brightness of ground = 1.23 c. per sq. ft.
V
BRIGHTNESS OF B
BRIGHTNESS OF AL
Brightness of AR = .695 c. per sq. ft.
Brightness of ground = 1.23 c. per sq. ft.

Fig. 4 (Fry and Bartley). Graph IV shows the effects obtained when AR is equal to the brightness of the ground and AL is below. Graph V shows the effects obtained when both AL and AR are below the brightness of the ground.

When AL is black, A appears to be almost black and seems not to be affected by the fact that AR is considerably brighter. In other words, the light imparted to the right eye has very little effect upon the net brilliance. The dextrocular activity initiated by AR must be depressed in some way by the sinistrocular process corresponding to AL. Now AL, being black, does not stimulate its part of the left retina, and sets up no activity in the corresponding optic nerve fibers, and this inactivity in the left half of the visual apparatus cannot be said to depress activity arising in the right. Hence the depression must be attributed to the process subserving border contrast between AL and its ground.

are plotted in graph V in figure 4. The brilliance of A increases as the brightness of AL is increased from 0 up to the point where the brightnesses of AL and AR are equal, but beyond this point the brilliance of A is not increased by adding to the brightness of AL. In other words, lowering the brightness of AL below that of AR decreases the brilliance of A, but raising it above fails to increase the brilliance.

Rivalry between AL and AR takes place only when AL is above and AR below the brightness of the ground, or vice versa. When AL is black, AR white, and the ground gray, AR and AL appear alternately in consciousness. The intermittent disappearance of AR cannot be attributed to the blackness of AL, but rather must be attributed to the process subserving border contrast between AL and its ground.

Summary

1. Further evidence is presented in this paper that binocular vision gives an increase in brilliance over monocular vision. This fact is accounted for on the hypothesis that pathways from each pair of corresponding points in the two retinae converge upon a common pathway in the brain and that summation takes place.

2. Graham's finding that binocular vision gives no lower absolute threshold than monocular vision is shown to be compatible with this hypothesis.

3. The process subserving border contrast plays an important rôle in the integration of the two converging streams of impulses. In order to throw light upon this aspect of binocular vision, Fechner's paradox and several other binocular phenomena have been studied.

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RECURRENT VITREOUS HEMORRHAGE

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Within a period of twelve years, thirty cases of hemophthalmus were admitted to the Out-Patient Department of the Massachusetts Eye and Ear Infirmary, in approximately 170,000 new admissions for ocular affections. These excluded vitreous hemorrhage due to trauma, arteriosclerotic, diabetic, and renal retinitis, vascular hypertension, tumor, and other extraneous causes. The literature is reviewed.

Apart from trauma, blood may enter the vitreous in a number of conditions affecting the vessels of the retina, choroid, or ciliary body. Profuse retinal bleeding of whatever etiology may result in vitreous bleeding. The cause, when there is a known preceding or concomitant ocular or general disease, is usually obvious. However, there are cases of vitreous hemorrhage which occur in persons of apparently good general health who have not had any history of ocular disturbance. These present a more or less uniform, clinical picture characterized by massive hemorrhage, the frequent involvement of both eyes, and the tendency to serious complications such as retinitis proliferans and separation of the retina. A most thorough general examination including all the modern diagnostic aids often fails to reveal any conclusive etiologic factor. This type of spontaneous vitreous hemorrhage has been recognized for at least seventy-five years but it is only within the last two decades that most of these cases have come to be regarded by many as probably of tuberculous origin.

Von Graefe[1] (1855) is usually cited as being the first to recognize this condition as distinct from other types of hemophthalmus. However, MacKenzie[2], in his fourth edition of "Diseases of the Eye," published in 1854, devoted considerable space to the subject and even described a typical case occurring in a male of twenty years who had repeated vitreous bleedings for which no cause could be found. Early writers referred to these cases as "essential relapsing hemorrhages" (Scrini and Bordeaux[3]) and to the complicating retinitis proliferans as "idiopathic" (Weeks[4]). Panas[5] suggested the name "ocular epistaxis," owing to its appar-

ent similarity to the commonly occurring nasal bleeding of unknown etiology. Eales[6] in 1880 first described the clinical picture in detail and cases of this nature are called Eales's disease in the British literature.

Eales studied five cases of recurrent vitreous hemorrhage. He laid special emphasis on the occurrence in adolescent males of epistaxis, constipation, bradycardia, dyspepsia, headache, lassitude, and the absence of syphilis or of any other serious constitutional disease to account for the hemorrhages.

The typical clinical picture presented by cases which have been reported in the literature, is as follows: a person, usually a young male, suddenly loses the vision of one eye. Sometimes a history of periodic epistaxis and chronic constipation is obtained and occasionally a positive family history of tuberculosis. Routine ocular and general clinical examinations throw no light on the etiology. A diagnostic tuberculin skin test may be positive. Within a variable period, not infrequently as short as four to six weeks, the vision of the affected eye may be normal or nearly so. After several weeks or months, or even years, hemorrhages recur and eventually, both eyes are involved. With repeated bleedings absorption of the blood becomes slower and vision tends to diminish. Despite many recurrent hemorrhages, useful vision may be retained; on the other hand, one or both eyes may become partially or totally blind. Organization of the hemorrhage with the formation of retinitis proliferans is a characteristic feature and a separation of the retina may develop secondarily. A low-grade iridocyclitis may develop at any time during the course of the disease. In rare cases, glaucoma may develop and enucleation may be required.

Examination of the anterior segment of the eye is entirely negative and slit-lamp examination fails to reveal any signs of anterior inflammation.

A fundus examination of the affected eye is usually impossible for some weeks following the occurrence of vitreous hemorrhage. The blackish appearance of fresh vitreous bleeding as seen by ophthalmoscopy is followed by the gradual reappearance of the red fundus reflex and after a variable period any one of several possible fundus lesions may be detected. An outstanding feature is the frequent involvement of the retinal vessels, especially the smaller peripheral veins, as evidenced by unusual tortuosity, engorgement, constrictions, dilatations, or the formation of varices (Friedenwald[7], Adler[8]). Retinal hemorrhages and sometimes a preretinal hemorrhage are visible. These may be small or large, round or irregular, are not flame shaped, and are generally found in the periphery of the fundus. Some cases present perivascular changes in the form of periphlebitic areas or of a perivasculitis (Axenfeld and Stock[9], Davis[10], Knapp[11]). In the former, whitish or yellowish, sharply circumscribed areas are found lying in direct relation to the retinal veins; in the latter, whitish bands are observed to run alongside the vessels. Choroidal exudates and foci of chorioretinitis may be found. The optic disc is generally not involved, though Gilbert[12] has described an uncommon form in which the optic nerve and vessels near the disc are primarily involved and this form may resemble the clinical picture of thrombosis of the central vein (Safar[13]). In late stages, bands of whitish connective tissue course from the retina in the vicinity of the disc into the vitreous, constituting retinitis proliferans, and a separation of the retina, resulting from the traction of the cicatricized connective tissue, may be evident.

Careful examination of the unaffected eye through a dilated pupil, in many cases fails to reveal any abnormality. In others the peripheral fundus may show changes of a nature above described; for example, in the course and caliber of the veins, retinal hemorrhages, and perivascular changes. Wilmer[14] has observed in the unaffected eye "a striated appearance of the retina with darkness of the venous current (sometimes with a white reflex from the vessel). The situation suggests an infiltration of the fluid por-

tion of the blood into the retinal tissue."

All writers agree that these cases are rare. Statistics as to incidence of occurrence are meager. Eales states that he had not observed a single case among 12,000 clinic patients. Nieden[15] mentions having observed six cases in young men in 34,489 eye patients. Scrini and Bordeaux[3] report the incidence as one in every 5000 or 6000 eye patients. Aubineau[16] found 8 cases among 25,000 eye patients. Finnoff[17], in 1921, collected a total of 110 cases reported in the literature and Young[18], in 1929, collected another 72 cases reported since 1921.

That the condition shows a marked predilection for males was early recognized and has never been contraverted. The belief, originally expressed by Eales, that normal menstruation acts as a safeguard against spontaneous vitreous hemorrhage is generally accepted. The occurrence of spontaneous retinal and vitreous hemorrhage in females has sometimes been observed in association with some irregularity of menstruation or sexual maldevelopment (Loeb and Meyer[19]). Of the 132 cases collected by Finnoff and Young, in which sex was specified, 108 were in males.

Race and heredity appear to play no role.

As to age, cases of this condition are usually referred to in the literature as "recurrent vitreous hemorrhage in adolescents" or "juvenile vitreous hemorrhage." The very great majority of the reported cases had the onset of the hemorrhages in the second and third decades of life. Cases have been reported to occur as late as in the seventieth and as early as in the tenth year of life.

Eales offered the following explanation to account for the hemorrhages:

> From the absence of any evidence of the various conditions known to cause hemorrhage, from the absence of albuminuria, diabetes, gout, syphilis and of any high arterial tension, from the character of the hemorrhage, and from the evidence of local variations of circulation and from the slow pulse, constipation, flushing of the face, headache, and puffiness and discoloration of the eyes, I am inclined to attribute this combination of conditions to a neurosis affecting the circulatory and the digestive systems, leading on the one hand to partial inhibition of the muscular movements of the bowels, and to a vasomotor contraction of the vessels of the alimentary tract with inhibition of its secretory functions, thereby causing dyspepsia, constipation, malnutrition, and on the other hand to a compensatory dilatation of the systemic capillaries, especially those of the head, and in these cases, of the retina, causing overdistention of the venous system and systemic capillaries, with liability to rupture on the occurrence of any intensifying cause. Hence the headache, the epistaxis, the retinal hemorrhages, and the fullness and tortuosity of the retinal veins and temporal artery.

Hutchinson[20] (1881) attributed the hemorrhages to an "inequality of the circulation," either a heightened or diminished arterial tension in the nature of a vasomotor imbalance.

Woods[21] at a meeting of the American Medical Association, in 1911, was unable to offer any suitable explanation or therapy after an analysis of his own cases and of the opinions expressed by his colleagues.

The state of chaos that exists in this field is illustrated by the numerous factors that have been suggested at one time or another (even to the present day), as playing some role in the production of these hemorrhages: gout, oxaluria, phosphaturia, helminthiasis, indicanuria, constipation, errors of diet, toxemia, auto-intoxication, lowered blood-coagulation time, raised coagulation time, low blood pressure, high blood pressure, hemophilia, hyperthyroidism, thyroid insufficiency, nephritis and renal insufficiency, syphilis, focal infection, diminished blood calcium, endocrine dyscrasia, and tuberculosis.

The largest number of cases reported in the literature, especially those in recent years, has been ascribed to tuberculosis. Of a total of 182 cases (collected by Finnoff[17] and Young[18]), 66 were considered due to tuberculosis, 10 others as probably due to tuberculosis, and 68 are tabulated as undetermined.

Simon[22] (1896) first called attention to tuberculous disease of the retinal vessels as a cause of intraocular hemorrhage in young people and Harms[23] first demonstrated the existence of tuberculous periphlebitis as a pathologic

entity. In 1909 and again in 1911, Axenfeld and Stock[24,9] suggested that many of these cases may be of tuberculous etiology, resulting from the focal action of the toxins of the tubercle bacilli on the retinal vessels in patients who are the subjects of a latent tuberculosis. Fleischer[25] in 1914 supported Axenfeld and Stock's contention and presented pathologic evidence which he considered proof that the hemorrhages resulted from the lodgment of tubercle bacilli within the walls of the retinal veins rather than from a tuberculous toxemia.

In 1912 Igersheimer[26] reported three cases of vitreous hemorrhage and confirmed the statement that tuberculosis is often the cause of disease of the retinal vessels and of its consequences such as hemorrhage into the vitreous.

In 1913 Knapp[11] reported four cases illustrating "Retinal changes in adolescents" and concluded that while the relation to tuberculosis was difficult to prove, a tuberculous origin in these cases seemed probable.

In 1916 Jackson[27] reviewed the subject of "Tuberculosis of the retina, recurrent hemorrhage, and retinitis proliferans" and stated:

> On the well recognized association of intraocular hemorrhage and subsequent connective tissue formation constituting retinitis proliferans, we have the same association of pathologic processes. The few cases that have been studied anatomically, the response of a large number of cases to the specific tuberculin test and the relative recoveries that are now recorded to the credit of the recognized treatment for tuberculosis, give sufficient basis for the view that tuberculosis chiefly of the retinal vessels is the essential nature of the clinical condition represented by the above cases of recurrent retinal hemorrhages in young persons followed by retinitis proliferans.

In 1912 Davis[10] presented a case featured by repeated vitreous hemorrhages and marked perivasculitis affecting mainly the retinal veins and in 1920 another featured by recurrent retinal hemorrhages and a marked involvement of the optic nerve. He regarded both as tuberculous on the basis of positive family history and positive tuberculin reaction. He stated in his last paper that "the weight of opinion, based on clinical and experimental observation together with a tuberculin-reaction test and the few pathologic reports that have been made, leans heavily to tuberculosis as a chief cause."

From a study of the literature and a presentation of five cases, three regarded as tuberculous and two as due to focal infection, Finnoff concluded that "recurrent hemorrhage into the retina and vitreous is probably not a specific disease" and that "tuberculosis of the retinal vessels, especially the veins, is one of the common etiologic factors."

Wilmer[28] (1928) classifies cases of recurrent hemorrhage into the vitreous as a definite type of tuberculous intraocular inflammation. "For the past ten years in every case of this kind, save one, where I have been able to make a survey, I have found the process tuberculous." Furthermore, he states, "Tuberculous eye lesions, especially those cases of recurrent vitreous hemorrhages in young people are often accompanied by evidences of subnormal bodily health—clammy extremities; pasty complexion; low blood pressure, temperature, and basal metabolic rate; slight general leucocytosis; occasionally lowered blood calcium content; and often general lassitude and depression."

Carmazza[29] (1928), reporting thirteen cases observed at the eye clinic at the University of Rome, agrees that tuberculosis enters into consideration as the most common cause of these hemorrhages but does not believe that every case should be regarded as tuberculous. He claims that relations can be established between tuberculosis and the coagulability of the blood, such that a temporary or persistent hemophilia is produced presumably by the action of tuberculous toxins on the blood mixture and on the vascular constituents.

Wurz[30] (1930) observed six typical cases two of which he diagnosed as tuberculous with certainty, and four of which he diagnosed as tuberculous with probability.

Motegi[31] (1931) observed a lengthening of the blood-coagulation time in six cases of juvenile vitreous hemorrhage.

Hutchinson[32] (1932) concludes from a study of the subject and a report of five cases that "there is no satisfactory evidence that tuberculosis, either local or remote, is a cause," and he suggests that the condition may be the result of some abnormality of the blood or some toxic product which we are unable to detect by our present diagnostic tests.

Syphilis has been mentioned as a causative factor in some cases. Igersheimer[26] mentions it as a rare cause and Finnoff[17] as an occasional cause. Only six of the 182 cases collected by Finnoff and by Young[18] were ascribed to syphilis. Three patients among the thirteen of Caramazza[29] had syphilis. Davis[10] suggests that syphilis and tuberculosis may coexist as etiologic factors. In a case of vitreous hemorrhage in a young woman of 24 years presented by Nethercut[33] before the Chicago Ophthalmological Society, Suker in discussion declared that it was most likely tuberculous in spite of the fact that the patient's blood gave a four-plus Wassermann reaction.

Godwin[34] has reviewed the subject as regards focal infection. Cases ascribed to diseased teeth and tonsils and sinus diseases have been reported by many. Godwin reports in detail in the case of a man aged 29 years, who had had a chorioretinitis and hemorrhage 8 years previously; a uveitis which cleared up after the extraction of teeth; chorioretinitis and hemorrhages following an acute tonsillitis; and, finally, repeated retinal and vitreous hemorrhages that ceased after a sinus operation.

Young[18] suggests that calcium deficiency may be a possible cause in some cases. In two of three cases studied, the blood calcium was lower than normal, and one seemed to respond to calcium therapy.

In 1920, Zentmayer[35] first stated that he did not believe that tuberculosis was entirely satisfactory as an explanation of the symptoms in these cases and suggested that some disorder of the endocrine glands, possibly the adrenals, might exist independently or coexist with tuberculosis as an etiologic factor. In a discussion of Young's paper, he expressed the view that the finding of a lowered blood calcium might have some relationship to an endocrine dyscrasia.

In cases of recurrent vitreous hemorrhage in females, associated with abnormal menses or occurring as a form of vicarious menstruation, it is hard not to think that a direct causal relationship exists between the two factors and that some disturbance of ovarian secretion, or abnormal sensitivity of the retinal blood vessels, or both, are at play.

Pathological examination has not been very enlightening in determining the etiology, as eyes afflicted with this condition are very seldom removed, and then only at a late stage, generally due to a complicating glaucoma. Cords[36] groups cases of retinal periphlebitis, recurring vitreous hemorrhages, proliferating retinitis, and retinitis with massive exudation (Coats' disease) under the heading of "Juvenile Retinal Angiopathy." The principal pathologic change in retinal periphlebitis is found in the walls of the retinal veins in which an infiltration of round cells, sometimes with epithelioid cells and giant cells, is observed.

Fleischer presented the following pathologic evidence to support Axenfeld and Stock's contention:

> A male, 36 years of age, affected with pulmonary tuberculosis, first had periphlebitis and retinal hemorrhages and choroidal patches in the left eye. The anterior segment of the eye was uninvolved. The hemorrhages cleared up and subsided. One year later, the right eye became involved with nodular iritis, retinal hemorrhages, and marked periphlebitis, and the eye was removed because of a complicating glaucoma. Microscopic examination revealed that the changes in the anterior part of the eye as well as the changes about the retinal veins were of a tuberculous nature. "Typical groups of epithelioid and giant cells, in places showing slight necrosis, were present forming nodules lying on one side of the vessels or sheaths surrounding them."

Safar[13] emphasized that secondary glaucoma is a not infrequent complication of recurrent vitreous hemorrhages, and reviewed the pathologic pictures presented by two cases in which the eyes were removed because of it. He

found that the glaucoma was caused by closure of the angle of the anterior chamber and the formation of a membrane on the anterior surface of the iris. In addition to the usual findings of a tuberculous phlebitis, he mentioned tuberculous leptomeningitis of the optic nerve, retrobulbar infiltrations in the septa of the nerve, and ciliary perineuritis as rare findings.

In a case recently reported by Clapp[37] in support of the tuberculous etiology of cases of spontaneous retinal and vitreous hemorrhages and of tuberculin therapy, one eye was enucleated after sight had been lost from chronic uveitis. Microscopic examination showed the process to be tuberculous. This fact plus the positive tuberculin test was accepted as proof that the recurrent hemorrhages in the other eye were of tuberculous origin, and, incidentally, under proper tuberculin therapy the process cleared up entirely.

Tubercle bacilli have never been isolated from the retinal lesions.

Attacking the problem of tuberculous etiology from an experimental viewpoint has not lead to any fruitful results. Intravenous injection of virulent tubercle bacilli into the rabbit produces regularly, according to Stock[9], a tuberculosis of the uvea but has never presented a localization in the retina. While it has been found impossible to produce retinal changes without lesions in other parts of the eye, the fact that a tuberculous periphlebitis as well as a uveal tuberculosis is produced when bacilli are injected into the common carotid artery of rabbits is offered as proof that the bacilli can accumulate in the walls of the retinal veins (Finnoff[38], Ito[39]). On the other hand, Spanlang[40] has pointed out that the picture in the retinal veins is not necessarily tuberculous, for he has produced similar perivascular retinal infiltrations in animals by repeated injection of blood into the vitreous.

Those who regard these cases as tuberculous in origin believe that tubercle bacilli are carried through the blood from tuberculous foci in other parts of the body directly to the perivascular lymph spaces where they lodge in the walls of the retinal veins and form specific nodules round the vessels, and that in the exceptional case the bacilli are carried first to the uveal tract and then via the lymph stream to the retina. (Axenfeld[41], Safar[13], Suganama[42]). Meller[43] and Frank[44] maintain, however, that the retinal pathology is always secondary to primary tuberculous lesions in the uveal tract. The actual hemorrhages are ascribed to localized pathologic weakening of the blood-vessel walls with resulting rupture. The newer conception that ophthalmic tuberculosis is in many instances the result of an allergic response to tuberculo-protein could well be advanced in cases of spontaneous vitreous hemorrhage.

Cases of spontaneous vitreous hemorrhage are entitled to a thorough medical survey. The possibility of the entrance of an intraocular foreign body unknown to the patient, or of the presence of an intraocular neoplasm should be borne in mind. A detailed history with special emphasis on the question of suspicious symptoms of tuberculosis and blood disease should be taken. Pathologic tests should include a complete study of the blood (count, serology, calcium content, bleeding and coagulation time), and a tuberculin diagnostic test; an x-ray picture of the chest should be taken in every case. A study with reference to the evidence of endocrine disturbance may be advisable. The ocular examination should include in addition to other observations of the sound eye, a careful fundus examination through the dilated pupil, for sometimes the earliest signs of disease are found peripherally.

The diagnosis of tuberculosis has frequently been made on the basis of one, or a combination of the following factors: Positive family history, positive lung findings, positive diagnostic tuberculin test (positive local and sometimes inadvertently positive focal reaction), the exclusion of other possible factors, and good results following the usual treatment of tuberculosis and tuberculin therapy. The presence of syphilis or focal infection does not exclude tuberculosis from considera-

tion as a possible causative factor. As to the tuberculin test, properly performed, most ophthalmologists regard it as an important diagnostic aid in the diagnosis of ocular tuberculosis.

It is necessary to differentiate the class of cases under discussion from cases of syphilitic chorioretinitis, which sometimes gives rise to profuse vitreous hemorrhage. These bleedings occur late in the course of the infection; are often bilateral; take long to absorb, and induce a proliferating retinitis early; often cause separation of the retina, and vessel formation in the vitreous; and may occur in eyes which have previously been affected with chorioretinitis and vascular disease.

The association of spontaneous vitreous hemorrhage and iritis with Henoch's purpura has recently been pointed out by Benedict[45] and Love[46].

It is difficult to estimate the ultimate outcome of an individual case, as the courses of observed cases have been so variable, but one can give a good prognosis with reasonable assurance as to restoration of vision at the onset of the disease. Early vitreous hemorrhages absorb very rapidly as compared with other types of vitreous bleeding. Meller[47] states in his discussion on the absorption of vitreous blood, "the most favorable condition obtains in otherwise healthy eyes in which the hemorrhages are due to a trauma which affects only a circumscribed spot without involving the other tissues of the eye, or where a spontaneous bleeding occurred in circumscribed phlebitis." On the other hand, the later hemorrhages rather tend to become organized with the resulting formation of a proliferating retinitis and a secondary separation of the retina. Loss of vision is the result of these complications as well as the associated retinal hemorrhages and the unabsorbed vitreous opacities. The retention of some degree of useful vision in patients who have suffered many hemorrhages and have had a generally stormy course has been frequently observed (Sattler[48], Friedenwald[49], Davies[50]). Jackson[51] and Lloyd[52] stress the general bodily resistance as being the determining factor in the final outcome. Lloyd states that it is not at all unusual for complete recovery to occur in young persons with good resistance without any special treatment, while the most pitiable cases occur in those with poor general resistance. Finnoff believes that cases occurring in females are less severe than those in males. Schieck[53] states that if one succeeds in bringing patients through the juvenile age without severe retinal damage, hope increases with the years that recurrence will cease and useful vision be obtained.

The indications for treatment are first to hasten absorption of the hemorrhage and second to prevent recurrence. The tendency to spontaneous absorption of the hemorrhage, at least in the early stage and even without any particular treatment, together with the inability to establish a conclusive etiology in many cases, makes it difficult to discuss therapy. The usual nonspecific measures, considered of value in any form of intraocular bleeding, may be instituted: complete rest in bed for a time; forbidding the use of the eyes, even to fixation with a double eye bandage; the application of local heat; careful sweating by the use of hot packs and hypodermic injection of pilocarpine; the subconjunctival injection of hypertonic saline solution or dionin solution; and the oral administration of alternative drugs such as potassium iodide or Fowler's solution, are measures which may be of value. Hamburger[54] has recently advocated the use of intraorbital cautery in vitreous hemorrhage. In every case, regardless of etiology, an attempt should be made to increase the general bodily resistance. Foci of infection, whether regarded as causative or not, should be eliminated. In suspected tuberculous cases, the usual measures employed in the treatment of pulmonary tuberculosis should be instituted either at home or, preferably, in most cases, at a sanatorium.

Tuberculin therapy has many advocates in this class of cases but it is difficult to estimate its value on any truly scientific basis. It should be given only by those who fully understand the theoretical and practical principles of its

usage. Improper dosage may result in consequences disastrous to the eye. The initial dose must be very small and subsequent doses must be graded according to the degree of sensitivity. Schieck[53] stated that he had never observed any untoward results from careful tuberculin therapy in cases of recurring vitreous hemorrhage and he warns that with this form of treatment, one should not expect to prevent recurrences due to new foci but only to heal the old foci.

The use of salvarsan for nonspecific therapy has been advocated by Benedict[55].

Autohemotherapy has been suggested by Gonzalez[56]. Wurz[30] used tuberculin therapy in association with some form of nonspecific therapy such as autohemotherapy or milk injections.

The production of anaphylactic shock, by the use of diphtheria antitoxin, has been advocated by Aubineau[16] and Candiotti[57].

Favorable results from the administration of thyroid extract have been reported by Angelucci[58], Bennett[59], Zentmayer[35], Sédan[60], and Arloing and Rainard[61]. In the case reported by the last named, which they ascribed to thyroid insufficiency, there was no recurrence of hemorrhage during continued thyroid therapy but a relapse occurred with its suspension.

Calcium therapy seemed to be beneficial in one case reported by Young[18], in which a low blood calcium was noted. Parathyroid extract and parathyroid hormone were administered subcutaneously to increase the calcium content of the blood.

Various procedures have been suggested concerned with the removal of diseased vitreous in advanced cases where there has been no fresh bleeding for some time. Spanyol[62] believes that vitreous aspiration is of benefit in these cases. Elschnig[63] advises the substitution of sodium chloride solution for the aspirated vitreous, and Braun[64], who collected the results of this procedure at Elschnig's clinic, has emphasized its value particularly in cases of spontaneous hemorrhages and juvenile bleedings.

As a last resort in cases in which one eye is totally blind and the other eye appears to be similarly threatened, ligation of the common carotid artery has been suggested. Mayweg[65] has reported a successful result, Axenfeld[66] a partially successful result, and Derby[67] a failure with this radical procedure. Recently Hambressin[68] reported a case in which ligation of the common carotid artery combined with x-ray therapy to the spleen and eye served to prevent further hemorrhage on the side ligated, while the other eye had more than ten hemorrhages. Repeated venesection combined with long rest in bed has been advocated by Kyrieleis[69] as a beneficial and less formidable procedure than common carotid ligation, and more recently Hamburger[70] has called attention to this form of treatment.

Excluding cases of vitreous hemorrhage due to trauma; arteriosclerotic, diabetic, and renal retinitis; vascular hypertension, intraocular neoplasm, and intracranial hemorrhage, and those following intraocular operations, 30 patients with hemophthalmus were admitted to the Out-Patient Department of the Massachusetts Eye and Ear Infirmary within the period of twelve years, 1920 to 1932 inclusive, out of a total of approximately 170,000 new eye cases. Of these cases, nine have had recurrent hemorrhages and 21 are recorded as having had a single hemorrhage. Most of the latter group were seen but once in the clinic or were under observation for only a short period of time.

Details pertinent to the history and examination of the first group are shown in the accompanying table.

Considering the group of 21 cases which are recorded as having had a single spontaneous vitreous hemorrhage, 16 were males and 5 were females. The right eye was involved in 15 cases; the left eye in 6 cases.

The age distribution was: 10-20 years, 3 cases; 21 to 30 years, 5 cases; 31 to 40 years, 8 cases; and 51 to 60 years, 1 case.

In fifteen cases the general clinical examination was negative; one of these was a case of hereditary lues with a strongly positive blood Wassermann.

TABLE. HISTORICAL AND PHYSICAL DATA ON AUTHOR'S CASES.

No. and Case	Admitted to O.P.D.	Sex	Family History	Epistaxis	Constipated	Age at 1st Hemorrhage	Eye first Affected	Other Eye Involved?	Number of Hemorrhages	Retinitis Proliferans	Separation of Retina
1. (J.S.)	1920	M	negative	no	no	45	O.D.	yes, 5 yrs. later	15+	O.S.	—
2. (M.G.)	1922	F				39	?			O.U.	O.U
3. (A.L.)	1924	M	negative	+	yes	28	O.D.	yes		O.S.	
4. (F.M.)	1924	M	sister pulm. T.B.			35	O.D.	yes, 2½ yrs. later		O.D.	
5. (P.C.)	1927	M	negative	"frequent" (+)	no	41	O.D.	yes			
6. (H.M.)	1927	M	negative	"frequent since childhood" (+)	no	18	O.D.	no	5	O.D.	
7. (W.I.)	1929	M	Mother pulm. T.B.	no		33	O.D.	yes, 2 yrs. later	[redacted]	O.U.	O.U.
8. (A.M.)	1929	M	negative	"recurrent up to 3 yrs. ago"	no	33	O.S.	yes, 10 mos. later		O.U.	O.U.
9. (J.B.)	1932	M	negative		no	26	O.S.	no	2		

TABLE. HISTORICAL AND PHYSICAL DATA ON AUTHOR'S CASES. (*Continued*)

No. and Case	General Physical Examination	Pulse	Blood Pressure	X-ray of Chest	Hematology	Wassermann (Blood)	Blood Calcium	Tuberculin Test	Foci of Infection	Last Recorded Vision (Corrected) O.D.	O.S.
1. (J.S.)	negative	86	130/80	negative	normal	negative	9.37	++ (Von Pirq.)	Diseased tonsils?	faulty light proj. (1932)	20/70−1
2. (M.G.)	negative		138/80							No record	
3. (A.L.)	negative	82	110/72	negative		negative		+ (Von Pirq.)	None	20/20 (1927) O.U. "much improved" (1932)	20/100
4. (F.M.)	pulm. T.B.			chr. fibr. phthisis		negative			chr. sinusitis.	20/30 (1927)	20/70
5. (P.C.)	negative	116	143/98			negative			none	20/200 (1927)	20/100+1
6. (H.M.)	negative	96	112/80	negative	normal	negative	11.24		none until 1932, abscess of tooth	20/20 (blurred) (1932)	20/15
7. (W.I.)	negative	80	120/76	negative				+ (Mantoux)	diseased tonsils	"Blind" (1932)	"3 percent normal vision"
8. (A.M.)	negative	70–80	135/80	negative	normal	negative		+ (Mantoux)	pyorrhea	20/30−2 (1931)	F. at 2½ ft.
9. (J.B.)	negative	88	115/75			negative			chr. tonsillitis	20/20 (1932)	Divergent eye good L.P.

The other six cases have no record of a medical examination.

Fundus examination of the uninvolved eye was recorded as negative in seven cases, six of which were viewed through a dilated pupil. In one case the uninvolved eye showed fine vitreous opacities, retinal hemorrhages temporally, a thrombosis of the superior temporal vein, and "paraphlebitis retinalis adolescentium." In the remaining thirteen cases there is no record of the examination of the uninvolved eye.

A further analysis is impossible owing to insufficient data.

Three of these cases came under my observation during 1932 and it is impossible to state whether they will fall into the class of recurrent vitreous hemorrhage. General clinical examination was negative in all. Chest x-rays in all were negative. Blood Hintons were negative except in the case of the male of 19 years with hereditary lues. Diseased teeth were present in two of the three cases. Blood studies of the three were normal and the bleeding and coagulation times and tourniquet tests were normal. Blood calcium estimations in each case were within normal limits. Endocrine disease was excluded clinically and the basal metabolic rates were —2, —13, and 12. All gave marked positive reactions to intracutaneous tuberculin injections (1/100,000 in two cases and 1/30,000 O.T. milligram in one case).

Summary

1. Recurrent vitreous hemorrhage is a rare syndrome, usually occurring in apparently healthy males in the second and third decades of life, characterized by the absence of trauma, by sudden onset, massive vitreous hemorrhage in one eye, the tendency to recurrences at variable intervals involving both eyes, and to the development of a proliferative retinitis and secondary separation of the retina.

2. Cases of recurrent vitreous hemorrhage may be considered as falling into one of two groups: a smaller, characterized by the presence of perivascular changes in the fundus in the form of a periphlebitis or perivasculitis, in which tuberculosis without question is the etiologic factor, and a larger, characterized by vitreous hemorrhage with or without retinal hemorrhage and by the absence of perivascular changes, in which the etiology has not been proved to be tuberculous. The high degree of sensitivity to tuberculin as shown by numerous cases reported in the literature and exhibited by the cases studied at the Massachusetts Eye and Ear Infirmary, serves to confirm the widely accepted view that tuberculosis directly or indirectly plays a dominant etiologic role. Blood disease, syphilis, endocrine dysfunction, and focal infection may easily be excluded from consideration in most cases as etiologic factors. The occasional findings of deviations from the normal which have been reported in the literature, such as low blood pressure, lowered calcium content of the blood, and altered blood-coagulation time, may be regarded as incidental findings of no real significance.

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ENTOPTIC PHENOMENA OBSERVED IN MY EYES BEFORE AND AFTER CATARACT OPERATIONS

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This report, of special interest to the psychologist and physicist in ophthalmology, will hold the attention of the clinician who hears the attempts of patients to explain similar phenomena, which are here described by a trained observer and clearly illustrated. This is an abstract prepared from a thesis on this subject which is offered in partial fulfillment of the requirements for the degree of Master of Arts from the University of Texas.

Introduction

The data here presented were recorded beginning a few days after I was abruptly informed that I had cataracts in both eyes and would be blind in two years. This was all the more trying to me because my mother had been slowly going blind for thirty years from the same cause. It was some relief when I remembered that I could see floating bodies in my eyes when using a compound microscope and therefore could probably see the cataracts and make observations of their progress. Later I accidentally found that I could get excellent images by using a tiny drop of water on my glasses, the focus being obtained by moving back and forth in front of an electric light. Some of these drawings were lost in the civil war in China in 1927 but enough were saved to make a fair report.

When I came to China in 1924 I had no signs of cataract and wore the following corrections:

Distance, Right eye, —7 D. sph. ⊂ 3.25 D. cyl. axis 175°

Distance, Left eye, —7 D. sph. ⊂ 1.75 D. cyl. axis 15°

On my return in 1927 I had to use the following corrections and even then saw very little:

Distance, Right eye, —13 D. sph. ⊂ 2.0 D. cyl. axis 90°

Distance, Left eye, —13 D. sph. ⊂ 1.75 D. cyl. axis 90°

In October, 1927, I was operated on in New York City. Through no fault of the doctor's the right eye was lost through infection but the operation on the left eye was successful and seven months later I was able to take up college work without tiring my eye. Most of the experiments were done at the University of Texas but I have continued to record phenomena as they have appeared and recently I have made a set of observations at Central China College to note changes caused by the present condition of the retina.

Entoptic Observations

All entoptical images are produced by having in front of the eye, at less than its focal distance, a pin-point image of some bright light to furnish the divergent rays that will cause any obstruction in the line of sight to cast a shadow on the retina[1]; the closer the point of light the larger the image. The compound microscope, the water drop, and light reflected from the lens in one's spectacles furnish this point of light. After one becomes adept in making observations, I think the last method the easiest. Without using a lens I have been able to get images of the hyaloid remnant, produced by the cornea. Internal reflection seems to be produced between the cornea and the covering of the vitreous when light enters at an angle from the upper left side. Figures 1 and 2 are sketches of the cataracts in their chronological order. The two upper drawings were made in Wuchang, China, and this description accompanied the original drawings: "That on the left eye is a bit smaller and has less radiating lines. It is surrounded by a clear border not visible in the right cataract. Floating bodies are noticed in both eyes." The "water-drop" drawing done in February, 1928, was made for Dr. Wheeler not long before the cataract was re-

moved. The "sun-set" drawing of August, 1929, was made when I first became disagreeably aware of the growth of the secondary cataract. The 1930 drawings show the secondary cataract just before and after the discission. The last one is how the eye looks today. The filaments across the opening in the capsule are in the vitreous and were not noticed at first. The change in shape of the observed field from a circle to a spherical triangle and back to a circle was probably due to the fact that the retina was too irritated to register clearly toward its outer edge.

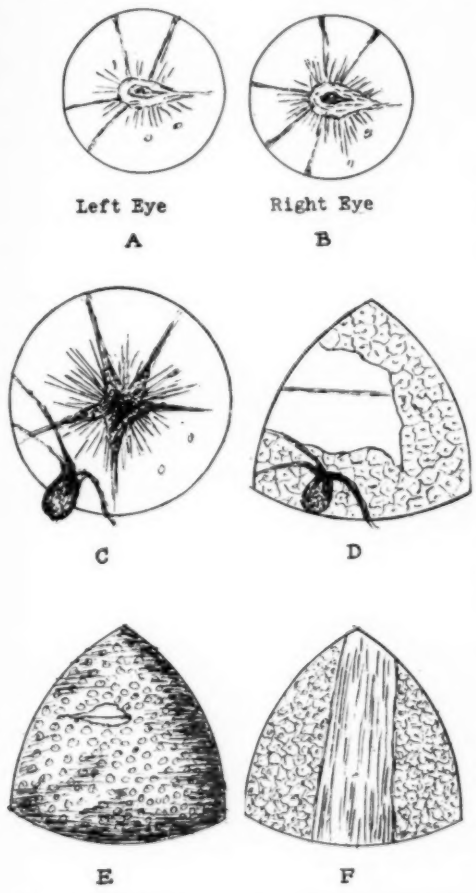

Fig. 1 (Clark). First views of the cataracts, June, 1926; A, left eye, B, right eye. C, just before the operation on the left eye, February, 1928; D, the secondary becomes noticeable, August, 1929; E, the secondary after a typhoid inoculation, April, 1930; F, soon after the discission, June, 1930.

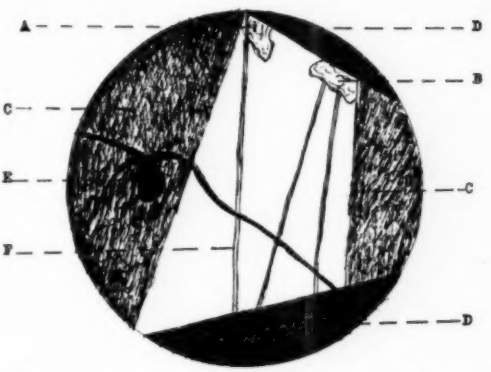

Fig. 2 (Clark). The eye as it appears today, November, 1932. A, fragment of the capsule; B, this protrusion is sometimes covered with what appear to be tiny, sharp, black crystals; C, capsule showing darkening by the secondary cataract; D, eyelids; E, hyaloid remnant; F, filaments in the vitreous.

The large floating body is a remnant of the hyaloid artery[2]. Mine is very inconvenient as it obstructs vision to a certain extent all the time and is particularly a nuisance when the button happens to be in the center of the field. It has changed position several times since I began to observe it. It reflects and refracts light and this is especially noticeable when monochromatic light is used.

When monochromatic light is used the muscae volitantes appear as small dark points surrounded by concentric circles produced by interference[3]. The eye-piece used to magnify the slit was not achromatic, causing the violet light to be focused farther from the eye than the red and the images were thereby decreased in size. With violet illumination the vascular figure can be seen. With the other colors there is too much diffraction to produce a clear image. It would be easy to imagine that these tiny particles might be corpuscles but for the fact that their motion is too slow and always downward. What Helmholtz considers effects from momentarily jammed corpuscles may be seen when looking at a bright sky. There are minute flashes of light which come in irregular lines similar to a network of capillaries.

Sketches of the above-mentioned phenomena may be seen in figure 3.

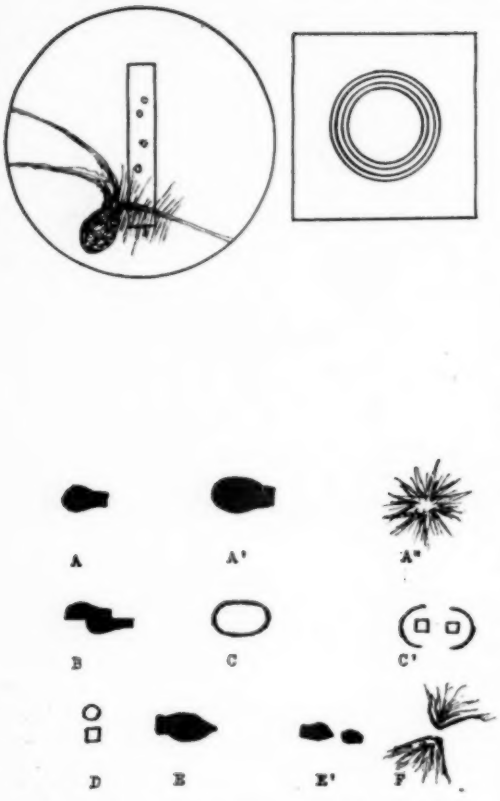

Fig. 3 (Clark). Above, a slit of monochromatic light shows diffraction rings produced by the muscae volitantes (detail of the pattern is shown in the square) and also shows how the hyaloid remnant obscures vision when it crosses the line of sight. Below, curious phenomena sometimes seen when the eyes are closed.

Photopsia

Both eyes have had periods of high tension and at such times I notice that after exercise I can see fiery rings that flash so brilliantly as to obscure vision until the phenomenon dies out. The flashes are synchronous with the pulse. There are flickers that are much faster and that break into two parts before they disappear from the field of vision on the nasal side. After the retina of my right eye pulled loose because of the infection I noticed several curious phenomena when the eye was closed. They are shown in figure 3.

"*A* is the most common form. It seems to start suddenly outside the eye, gets larger as it approaches, and finally disappears to burst at the back of the eye. Sometimes it divides into two parts as shown in *B* with sometimes the lower and sometimes the upper half displaced to the right." In each drawing black lines represent fiery lines in the eye. These drawings and descriptions were made for Dr. Wheeler and may be found in his history of my case.

In August, 1929, I saw a black after-image of a flicker obscure part of the colored circle which I can see in front of my blind right eye after having been out in a bright light. The violet and green effect first appeared after the enucleation of the right eye. At that time the bands were vertical as shown in *A*, figure 4. After a few months they became mere interminglings of the two colors. Horizontal bands appeared after the cataract was removed from the left eye but they soon disappeared. In the dusk the colored circle can be projected into space and objects seen through it. This phenomenon is probably caused by the inflamed condition of the retina. Pressure on the left eye causes a flash of light to appear exactly in the same position as the colored circle and obscures it entirely for the time being.

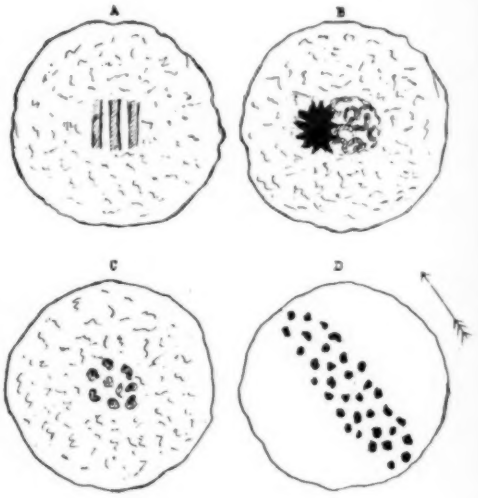

Fig. 4 (Clark). A and B, the two stages of violet and green bands which appeared in front of the blind eye. The black spot is an after-image. C, bright blue spots on a dark ground were seen in the left eye; D, a shower of pink spots passes over the blind eye with a sharp click.

Anything that affects the circulation causes color flashes. After taking quinine or aspirin alternate green and violet pulsations appear in about half an hour. Just before going to sleep there seems to be an audible click in the right eye which is followed by a shower of pink sparks on a dark-blue ground as shown in *D*, figure 4.

Ever since the operation my left eye has been extremely sensitive to green and violet and this may partly account for the results obtained in several experiments done in color.

Color Vision

In 1911 I could see only the first violet line in the mercury spectrum but in 1929 the violet was quite plain at 334 millimicrons and it was faintly visible at 330. At this time I was using a monochromatic illuminator built by Gaertner of Chicago and the spectrum was furnished by a hundred-candlepower Pointolite. This power to see so far into the violet was caused by the removal of the lens which absorbs a great deal of violet light[4].

I thought it would be interesting to repeat Dr. Frank Allen's experiments with the flicker spectrometer[5] since my eye was abnormally sensitive to green and violet.

I tried to repeat the experiments and plot the results as nearly as possible as Dr. Allen had done them. In the summarization of the results of his curves Dr. Allen says[5(c)]: "Where the curve obtained after stimulation is above the normal, it shows that the color has become physiologically less bright, and that the corresponding color sensation has become depressed in sensitiveness; where it is below the normal, it indicates that the color has become physiologically brighter than normal, and therefore the color sensation is enhanced." It would be foolish to draw many conclusions from the few curves that I made but my notes, which were made before the curves were drawn, show that just the opposite is true for my abnormal eye. In all cases except where green was the stimulating color, the violet shades become faded. When green is used for stimulation the violet shades become richer and more like those of an arc spectrum. Yellow always seemed brighter when a stimulating color was used and with the blue and violet stimulation it became painfully brilliant. All color stimulations cause the green shades to become brighter. I get the same definite inflections in the red, green, and violet parts of the curves that Dr. Allen does and he thinks this supports the Young-Helmholtz color theory.

Before discussing after-images I will state how the colors look to me. From 740 to 660 is red. At 640 is a clear orange while from 620 to 580 is yellow. At 570 the slit is filled with half yellow and half green light. From 560 to 540 I see green. At 520 the slit is again divided into two colors, showing green and blue. At 505 green and blue both appear in the slit. From 500 through 440 is blue. At 427.5 blue and violet appear together in the slit. When stimulated with other colors I see violet from 420 to 340 but when the eye is rested I can see violet as far as 330. After noting the places between other colors, where two colors were seen in the slit when the eye was stimulated with light of a certain wavelength, I tried to find a place of the same kind between red and orange or between orange and yellow but was unsuccessful. In September, 1932, I saw for the first time Dr. Ladd-Franklin's book, "Colour and colour theories," and was astonished to note that the wavelengths at which I saw two colors were the ones at what I would call the "critical points" of her version of the color triangle. I repeated the observations here at Central China College and was even more astonished to find that while I no longer saw the colors in pairs as before I now saw them in layers superposed on each other in a way that fitted in with the Helmholtz-König Curves which are published for the first time in this book. For instance, at 468 I saw green, blue, and red superposed in the order given. The green was on top of the blue and seemed to come from the violet end of the spectrum while the red was under the blue and was on the red end. After great fatigue the red

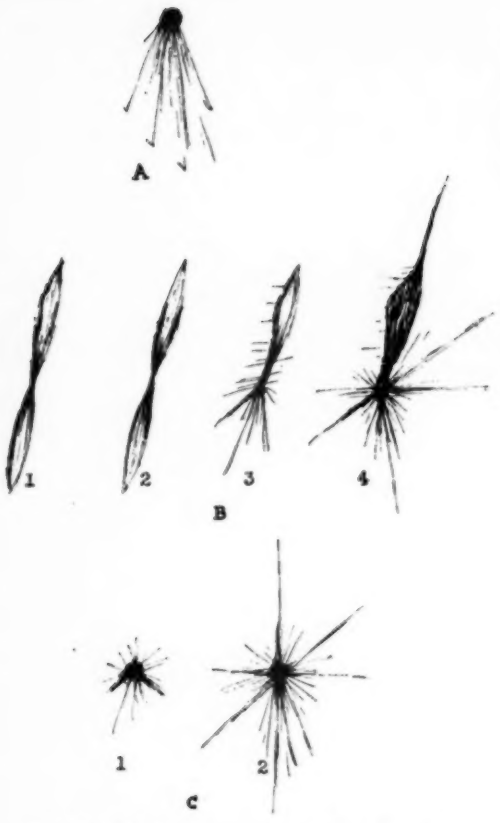

Fig. 5 (Clark). A, a street lamp looked like this after the iridectomy; B, stages in the development of "light tails" on a street lamp after the cataract operation; C, a lamp as it appeared when I first got my glasses and as I see it now.

and green changed places. At 409 red appeared under violet and two later observations showed green under violet or at both ends of the slit when the wavelength read 391.

After-images

In all cases of stimulation with monochromatic light, except the violet, at the end of twenty seconds there appeared around the slit a bluish-violet elliptical glow in the center of which the usual colored after-images appeared. These later faded to violet. Yellow alone gave a positive after-image. When violet is the stimulating color there is no preliminary glow nor final fading into violet. At those places where two colors were seen in the slit, the after-image showed two colors. Twice I have been able to refer after-images from the seeing eye to the blind one. At one time the black after-image of a flicker partly obscured the mingled green and violet so often seen in front of the blind eye.

Violet reflections

Before the discission for the secondary cataract was made I often saw above flowers and some other things a distinct violet haze that seemed to quiver much as heat waves above hot

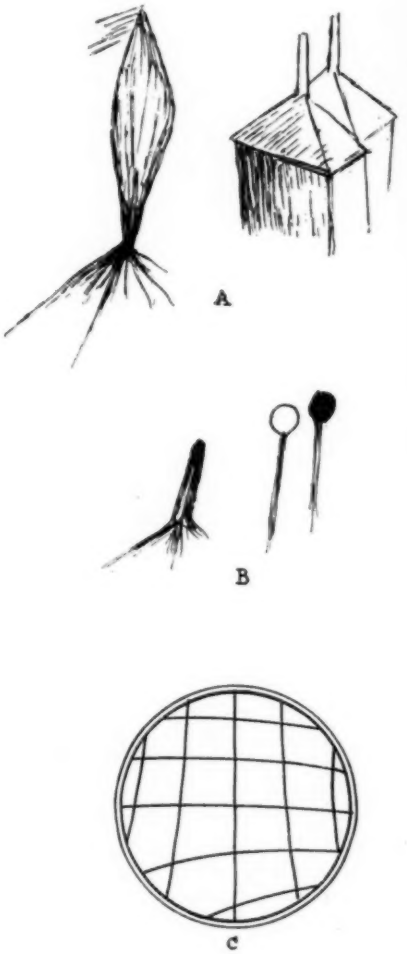

Fig. 6 (Clark). A, distant objects; B, objects only a few inches from the eye, distorted effects caused by astigmatism; C, aberroscopic drawing of the left eye, showing the surface of the cornea.

sand. I do not know whether this was due to uncorrected astigmatism or to the extreme sensitiveness of the retina. It was not visible above all objects and I never see it now.

Astigmatism

The removal of the lens left my eye with a most unusual amount of astigmatism which has not been reduced with time, as often happens. As a result, if I am not wearing my glasses all objects are distorted and a light becomes a long streak. Some of the queer effects are shown in the accompanying sketches. If the object is a distant one the distortion seems to go away from me but when the object is near by the distorted effect seems to come out toward me from the object. The aberroscopic drawing shows how the cornea is flattened.

Accommodation

With the removal of the lens all power of accommodation by that means was lost but I seem to have an unusual amount of accommodation which is evidently achieved by the muscles controlling the eyeball. When the light is good I can read any ordinary print, even though it be very fine, with my long-distance glasses but it tires me to do it long at a time. If I read any length of time I hook on a two-sphere lens over my eleven-cylinder, long-distance glasses. The correction of both the sphere and cylinder had to be increased after the discission for the second cataract.

Boone Compound.

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METASTATIC CARCINOMA OF THE CHOROID

Report of a Case

PERCE DELONG
PHILADELPHIA

Primary carcinomas of the choroid are unknown; the metastatic tumor occurs more frequently in the female than in the male, the primary seat in the former being rarely found in the uterus. In the case described the ocular condition brought the patient into the hospital where the eye was enucleated, the pathological report confirming the clinical diagnosis. Read before the College of Physicians, Section on Ophthalmology, in Philadelphia, December 15, 1932.

Many cases of metastatic carcinoma of the choroid have been reported, but its rarity warrants the citation of any additional authentic case, as each new case presents certain features of clinical and pathological value. Therefore, I would add the following case to the list already reported.

The most extensive studies of this condition published are the articles of Marshall[1], Parsons[2], Lagrange[3], and Oatman[4], and I refer the reader to these for detailed study. The work published conjointly by Suker and Grosvenor[5] includes a complete record of cases reported to 1909, and they add forty-seven cases, bringing the total then reported to sixty-four. In 1923, C. W. Usher[6] stated that one hundred and ten cases had been reported to that time. D. Michaïl[7], early in 1932, stated that the total number of cases reported to that year was one hundred and twenty-nine, and reported one additional case. In September, 1932, Foster Moore and H. B. Stallard[8] reported one case. These, with my case and cases reported by American authors, which were not included in Michaïl's total, bring the number to one hundred and thirty-five.

It is difficult to obtain accurate statistics of this disease, for it is reasonable to suppose that some patients in the last stages of cachexia die without the ocular lesions being recognized. Lagleyze[9] states that it occurs in one to one hundred thousand patients. Moorfields Hospital has recorded two cases in two hundred and seventy-six thousand eye cases. According to Michaïl, Uhthoff found a frequency of one in each thirty thousand cases, while Sattler found a lesser frequency of one in one hundred and fifty thousand. In opposition to this statistical research, Michaïl cites the opinions of such authors as Graeff, Clapp, Bouquet, Fehr, and above all van der Hoeve, who held that the condition was much more frequent than was demonstrated by statistics, because the ocular condition had no place in the diagnosis of a primary carcinoma, and the retinal detachment made it difficult to recognize. Van der Hoeve's conjecture is possible, but clinical records show that choroidal carcinoma is very rarely encountered. Michaïl states that in his examination of all the enucleated globes in his clinic over a period of eleven years, and in examining forty-eight thousand cases, he has encountered but one case of this kind.

In this discussion I restrict myself to specific cases of metastatic carcinoma of the choroid. Suker and Grosvenor state that: "A primary carcinoma of the endo-ocular tunics has up to the present day never been seen or reported, this for the reason that within the globe there are no histologic cells in which a carcinoma *per se* could engender." It is true that primary carcinoma of the choroid has never been seen nor reported, but within the globe there are histologic cells in which a carcinoma *per se* could originate. The retina with its pigment epithelium, the epithelium covering the ciliary processes, the pigment epithelium covering the posterior surface of the iris, the sphincter and dilator pupillae muscles, and the neurological and nervous elements of the optic nerve are all derived from neural ectoderm, and therefore are theoretically structures in which a carcinoma could develop. All carcinomatous involvements of the choroid as yet reported have been metastatic, but it must

be stated that several cases of primary carcinoma of the iris and ciliary body have been reported.

The usual seat of the primary growth (in the order of its frequency) is breast, lungs, stomach, liver, mediastinal glands and the thyroid. Rarer locations are the suprarenal body, prostate, and ovaries. The greater proportion of the patients suffer from carcinoma of the breast.

In this analysis it is worthy of note that although carcinoma of the uterus is of frequent occurrence, intraocular metastases therefrom are rare. Metastases in the uterus, however, from the primary organ are of quite common occurrence. In a study of the various statistics, no warrantable deduction can be drawn as to why metastases from the uterus should not be at least as frequent as those from other organs. A metastasis to the choroid can occur only through the blood vessels, since it is inconceivable that deposits can be brought from the breast and liver by way of the lymphatics, although cases have been reported in which the carcinoma cell was said to have reached the eye by way of the lymphatic channels.

The tumor is usually located in the posterior half of the globe, and is deposited there by an embolus. This is easy to explain because once the embolus enters the ophthalmic artery it follows preferably the ciliary branch, as this does not leave the artery at such an acute angle as the central branch; also the embolus follows the current of greatest velocity and usually occupies the center of the stream. The earliest focus is, in all probability, of the nature of an embolus in the finer arterioles of the choroid. The vessel affected is blocked and the cancer cells proliferate and burst through or replace the walls. They are then situated in the lymph spaces in the choroid, which lie directly outside the blood vessels and between the planes of the choroidal stroma—the direction of least resistance. For this reason most of their growth is lateral, a minimum increase in thickness taking place. This description applies only to the essential tendencies of development in given cases, which tendencies are frequently modified in detail by the proximity of vortex veins or other conditions, so that a new path of diminished resistance is open to the growth.

As the carcinoma grows in the line of least resistance it is usually confined to the first tissue invaded, for seldom do we find metastatic foci. As the tumor cells proliferate within the lymph spaces of the choroid, the appearance of the growth in its very earliest stages would be spherical, but because of the reduced pressure laterally in the lymph spaces, it will assume a disclike appearance. Later, the resistance on the side of the sclera makes itself felt, therefore the advance of the growth is less in this direction and the tumor becomes roughly elliptical. It grows twice as fast laterally as in thickness, for it is seldom thicker than two to three millimeters at the center. It finally encircles the disc, and cancer cells are often found penetrating into the intraneural lymph spaces. Bruch's membrane is seldom perforated. For this reason, and because of the early subretinal detachment, cancer cells are rarely seen in the retinal structure. This phase is contrary to that usually seen in sarcoma.

The tumor grows more rapidly toward the temporal side, and follows the line of least resistance, the lymphatics. It usually assumes the characteristics of the primary growth; i.e., medullary, scirrhus, or adenomatous. All three types may manifest themselves in an individual metastasis, but, as a rule, the type of the primary growth predominates.

It usually presents small hemorrhages within its structure, and they appear also in the choroid. Necrotic areas are frequently observed, recognizable by a yellowish color. The adjacent structures are involved by direct extension of the tumor cells. These cells rarely perforate the sclera, but when bulbar growths are manifested, they are due to metastases from the choroid via the ciliary vessels and nerves. Many of the above tendencies of carcinoma are the direct opposite of those of sarcoma.

In several instances metastases have invaded the other eye. Many writers

Fig. 1 (DeLong). Characteristic appearance of gross specimen.

express the view that this may be accomplished through the lymphatic channels by way of the optic nerve. If this were the case, the metastasis would manifest itself in the papilla. This, however, is not the case, so the only possible route for the extension would be by way of the posterior ciliary vessels. Generally speaking, carcinoma cells migrate preferably by way of the lymphatic channels, and one of the most striking features in the dissemination of the carcinoma is the readiness with which the lymphatic system becomes involved. This is borne out by the fact that the adjacent lymph glands are practically always the part to become affected, and it is only in the later stages that there is positive evidence of the vascular system having become the means of carrying the tumor cells to a remote part of the body. Consequently we should expect only a rare affection of the choroid and that in the last stages of the disease. This is the exact condition found in the majority of cases. However, Foster Moore and H. B. Stallard only recently published a case report in which they stated that the secondary growth developed eighteen years after the removal of the primary growth. Before this report was published the average span was less than one year. The average age of the patient is forty-five years, and the disease occurs more frequently in females, in a ratio of three to one.

The only other intraocular tumors for which metastatic choroidal carcinoma can be mistaken are the flat sarcoma and the so-called endothelioma. While both are of infrequent occurrence, the latter is extremely rare, and usually locates by preference in the ciliary body.

The case which I had the opportunity of studying pathologically was admitted to the Lankenau Hospital on the service of Doctor William T. Shoemaker. The data are as follows:

L.M., white, female, aged forty-eight years, was admitted to the hospital July 19, 1932. She complained of pain of

Fig. 2 (DeLong). High magnification showing proliferation of the epithelial cells, the connective tissue stroma and tuberculi, with adeno-papilliferous arrangement of carcinoma cells.

a "boring" character in the right eye, with disturbance of vision, distortion of objects, and "light flashes."

General examination revealed a well-nourished woman whose medical history was negative for heart, kidney, and lungs. The abdomen at this time was soft and relaxed and no tenderness nor mass was detected. A subsequent examination of the abdomen revealed a nodular mass in the upper right abdomen, under or attached to the liver.

There was some exophthalmus of the right eye. The pupils were equal and reacted to light and accommodation. The sclera was somewhat injected. The fundus examination was not satisfactory. The left eye revealed normal eyegrounds.

The clinical diagnosis was choroidal sarcoma. The eye was enucleated.

The patient was discharged July 25, only to return July 31. She complained at this date of a severe stabbing pain over the right lower ribs, describing it as a "stitch." This was exaggerated by motion. The pains had become acute two days before admission and were now exaggerated by deep breathing. She had no cough.

X-ray examination of her chest revealed a moderate bilateral diaphragmatic lag and a slight haziness over the left base. There was no evidence of demonstrable sarcoma at this examination.

The patient was discharged August 26 on an opium régime. She died two months later.

The pathological findings in the enucleated eye were as follows:

Macroscopically, the specimen, a globe of normal size, upon section revealed a shallow anterior chamber. In the posterior portion there was a neoplasm, flat in shape, of which the greatest anteroposterior diameter was 3 mm. It terminated rather abruptly at the equator. The overlying retina was detached by a subretinal serous exudate that separated the retina from the neoplasm.

Microscopically, the preëquatorial section of the globe showed slight evidences of edema with a secondary glaucoma, manifested by a small ciliary body and processes, which were dragged forward. There was an almost complete absence of the anterior chamber and complete obliteration of the filtration angle. The retina was detached and showed evidences of disorganization and early edema.

The neoplasm was composed of epithelial cells, having a tubular or adenoid arrangement. The arrangement of the epithelial cells was rather irregular in certain areas, while in other areas they had a cuboidal outline. Other cells appeared to be globular in shape. There were no areas of necrosis or hemorrhage. The advancing cells could be seen infiltrating into the lymph spaces of the choroid.

The stroma was composed of connective tissue with branching tuberculi. The choroidal vessels were distorted, but were present in the stroma. There was some new-vessel formation. Bruch's membrane was intact. There was no metastasis to the optic nerve.

Conclusions

1. Metastatic carcinoma of the choroid is much more frequent than is commonly supposed.

2. Primary carcinoma of the choroid is theoretically possible, but no case has ever been reported.

3. The metastasis is usually through the blood stream.

4. All types of carcinoma cells may be observed in the ocular growth, but that of the primary growth usually predominates.

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MEDICAL ECONOMICS

WALTER F. HOFFMAN, M.D.
SEATTLE

The writer foresees that the state will eventually take over the function of medical care for the sick, and forewarns his confrères that they must have a constructive program, satisfactory to the practicing physician, ready to be put into action when that day comes. President's address before the Twentieth Annual Meeting of the Pacific Oto-Ophthalmical Society at Seattle, June 30 to July 2, 1932.

Friends, fellow members, and guests of the Pacific Coast Oto-Ophthalmical Society, Seattle welcomes you to our Twentieth Annual Meeting. In welcoming you here I want to thank you and give due credit to the men who have contributed to the program; to Dr. Frederick Kiehle and the members of the other committees who have contributed unstintingly of their time, for the success of the meeting.

In the past years the President's address has embodied some original work, or has presented some message. I have no original work to present, and anything I might offer in the way of a message would be so trite as to be boresome. However, I cannot refrain from stressing the thought that has been running in the minds of all of us more recently, perhaps, than ever before.

The world at large, and the medical world in particular, is facing a changed economic situation. Values have been upset and human relations have been greatly disturbed, and we of the medical profession will encounter some of the greatest of these changes; and the question arises, "What new conditions will we have to accept, and who will mold and determine them for us?"

Will we fashion these changes satistorily for ourselves, or will we permit others to arrange our ways of conducting our business, and even our manner of living? I for one believe that we should do this for ourselves, or that some one among us, specially trained for this problem, should do it for us. We devise and perfect our scientific methods, medical, surgical, and laboratory. We discover and improve our remedial agencies; we invent or design our instruments; but we apparently are leaving to others the planning of our methods of carrying on our business. I know, and you know, that they are not planning greatly to improve the doctor's status, economic or social.

Many of us spend a part of each day hard at work in some clinic, where those who care to come, get the best we have, without remuneration to the doctor. These same people do not expect, nor would they ask for free barber service; free laundry service; free plumbing repairs; but they accept our best efforts as a matter of course, and as if it were their due without even a "thank you"; and this state of affairs is growing by leaps and bounds. The laity are being educated to expect and accept it.

In a recent issue of a nationally distributed magazine supplement of the Sunday papers, in an article entitled "How to marry on $35.00 a week," the author romantically labels her young couple Jack and Jill, and budgets their expenditures in a very interesting manner. After mentioning a few items, such as two daily papers at $18.00 a year; baseball games, fights, and movies at $104.00 a year; barber's bill and tips at $9.00; cigarettes $26.00, she then brings the doctor into the picture with this very naïve and illuminating remark: "If Jack or Jill are ill they must go to some clinic for treatment, as there will be very little money for doctor bills."

Again, to quote from an editorial in the Saturday Evening Post—this was just a line, but it struck me with tremendous force—the editor did not say, "If the practice of medicine is taken over by the State," or "unless the practice of medicine is taken over by State;" but he comes out very positively and says to a million readers "When the practice of medicine is taken over by the State." He is speaking of our present manner of practicing medicine, and leaves no doubt in the reader's mind

that it is just a matter of time before the State takes unto itself the function of caring for our ills.

Now all these changes are more or less a matter of education. In a recent number of the Journal of the American Medical Association, Dr. Morris Fishbein says, "People do not arrange to lay aside the funds that they could lay aside if they wished. *We* have been educating the public wrongly. They have not been taught to anticipate sickness. They have learned that death is inevitable, but they have not learned that sickness is also inevitable. They must learn that medical care is necessary, and they must save for it just as they now save for radios, cars, and burial funds." The medical profession has been educated to give a certain amount of free service; but the people are being educated daily to expect more and more for nothing.

I do not know that we can hope for some Moses to arise and lead us out of the wilderness, but there certainly exists some one of our profession, or some one who sympathetically understands our problems, who can offer a plan, or the beginning of a plan, on which we may unite, and which we may then pass along to our successors. I feel that we must give this as much thoughtful consideration as we do our scientific problems. We might listen to the advice of recognized economists, and make the matter a subject of a paper in our local and national societies; but we must not, like the ostrich, bury our head in the sand, or like the Christian scientists say with regard to physical ailments, "These things do not exist."

If, as is undoubtedly true, some change is to take place and we are to accept State Medicine, or some altered form of practice, we must begin this educational process. We have been striving to teach the world what the doctor can do, and have succeeded in no small way. I believe the people have confidence in our scientific ability, though they may belittle our business acumen. So the task remains to teach them that medical service costs money, and is worth it. However, we may carry on our profession, the laity should know, or should recognize a few fundamentals:

First. The old national motto "Millions for pills, but not one cent for preventive advice" must be junked.

Second. A medical education costs more and takes longer than preparation for any other profession; and the assurance of a livelihood in the practice of medicine should be sufficient to permit medical students time to prepare adequately.

Third. The remuneration should be high enough to permit doctors to keep up-to-date with medical journals, books, and postgraduate courses; to live in a decent manner, and have the vacations or rest periods necessary to such an active type of living.

It is our problem to instill these fundamental ideas into our own minds, and then into the minds of the lay public; so that if, or when, our business methods are reformed by or for us, the change will be for the better and not for the worse.

817 Summit avenue.

EXOPHTHALMOS IN LEUKEMIA

ALGERNON B. REESE, M.D., AND LOREN GUY, M.D.
NEW YORK CITY

In a case of lymphatic leukemia, the ocular manifestations first led the patient to seek medical advice. Exophthalmos is a rare symptom of myelogenous leukemia, but in the case reported it was induced by an expulsive orbital hemorrhage and was accompanied by choroidal hemorrhage with retinal detachment. A report of these two cases was presented at the meeting of the Section of Ophthalmology of the New York Academy of Medicine, December 21, 1931.

In the cases of leukemia that are seen by the ophthalmologist the commonest manifestations are retinal and conjunctival hemorrhages, puffiness of the eyelids, and a pale milky appearance of the fundus and its blood vessels. In 149 cases of definitely established leukemia (29 acute myelogenous, 64 chronic myelogenous, 24 acute lymphatic, and 32 chronic lymphatic), of which only a small number were examined by an ophthalmologist, retinal hemorrhages were noted in 9.3 percent, conjunctival hemorrhages in 4.7 percent, and puffiness of the eyelids in 2.5 percent. A less common manifestation is exophthalmos. In lymphatic leukemia, the records of 96 cases show that exophthalmos was present in 2 percent and many similar cases are reported in the literature. In myelogenous leukemia, on the contrary, exophthalmos did not occur in a series of 133 cases, and there is no mention of it in the literature.

The following case of exophthalmos complicating lymphatic leukemia is reported because the exophthalmos was the first manifestation of the disease, to prompt the patient to seek medical advice. A man, aged 69 years, applied for admission to Dr. Samuel's Clinic at the New York Eye and Ear Infirmary because his left eye was red and had become progressively swollen during the past month. He had lost 10 pounds and had felt below par physically for the preceding two months. There were no symptoms referable to the right eye.

Ocular examination: The right eye was normal, having a vision of 20/20. The left eye (fig. 1) presented an exophthalmos of 2 mm., with the globe displaced slightly downward. The bulbar conjunctiva was markedly congested and protruded like a sausage over the upper half, concentric to the limbus. This mass was uniform in contour and of a soft pliable consistence. The lacrimal gland was palpable. The upper and lower lids were thick and indurated, with an anterior convexity, as though pushed outward by a uniform infiltration in and posterior to them. Palpation of the orbital contents through them encountered resistance. The iris surface had a bombé contour and there was an old chronic iritis with posterior synechiae around the entire pupillary circumference. Tension was normal to palpation. The fundus appeared to be normal but the presence of some lens opacities prevented a clear view. Vision was 20/70. Although the existence of a lymphatic leukemia was not suspected, a biopsy of the protruding mass, showing tissue composed of a

Fig. 1 (Reese and Guy). Lymphatic leukemia with exophthalmos (left). Protrusion of bulbar conjunctiva and lids.

solid mass of lymphocytes, led to the correct diagnosis.

Physical examination: Generalized lymphadenopathy and enlarged spleen and liver.

Laboratory findings (Mr. E. B. Burchell M.Sc.):

White blood cells	160,000
Differential count:	
Small lymphocytes	94 percent
Large lymphocytes	1.8 percent
Polymorphonuclear leukocytes	3.8 percent
Eosinophiles	0.2 percent
Myelocytes	0.2 percent
Red blood cells	4,100,000
Hemoglobin	85 percent

The patient was transferred to the New York Hospital, where he received three exposures to radium. After this he was discharged and since then all attempts to locate him have failed.

Discussion. In cases of lymphatic leukemia there is a hyperplasia at the sites where lymphoid tissue is normally found, such as lymph glands, spleen, tonsils, etc. In the region of the eye, lymphoid tissue is present only under the conjunctiva and in the lacrimal gland. Examination in the case here reported gave the impression that the infiltration of the orbit was confined to the anterior portion immediately contiguous to the conjunctiva and lacrimal gland. In fact the striking feature was not the exophthalmos, but the protruding bulbar conjunctiva and indurated lids. This is the point which suggests a lymphatic leukemia as an etiological factor in exophthalmos. It is highly probable in this case that the mild exophthalmos was due, not to a pushing forward of the globe, but to an outward pull caused by a shifting *en masse* of its anterior adnexa.

In addition to the generalized lymphadenopathy in lymphatic leukemia, large localized tumor masses of lymphocytes occasionally appear at some site where lymphoid tissue exists; in the present case, for example, this occurred in the region of the eye. The following explanation has been suggested: A hyperplasia of lymphocytes occurs in the lymphoid regions of the body, and a sort of balance exists between their production by the germinal centers and their drainage by the lymphatics into the general circulation, with a preponderance on the production side. If, however, at some site the drainage is interrupted by a partial or total occlusion of the lymphatics supplying this region, the lymphocytes accumulate and form a large tumor mass. This, presumably, is what occurred in the case under discussion.

A normal blood picture does not necessarily rule out a potential lymphatic leukemia since the generalized lymphadenopathy and even a localized mass of lymphocytes may be present before the lymphocytes appear in abnormal numbers in the general circulation. Later such cases of pseudoleukemia almost invariably give a positive blood picture.

Exophthalmos complicating myelogenous leukemia is exceedingly rare. No cases are reported in the literature and Meller[1] stated that in his opinion this disease never showed orbital involvements. The following case is therefore reported.

A man, aged 19 years, was admitted to Dr. Shine's Clinic at the New York Eye and Ear Infirmary, complaining of sudden loss of vision in the left eye five days previously. There were no symptoms referable to the right eye.

Ocular examination: In the right eye vision was 20/30. The ophthalmoscope showed numerous hemorrhages in the nerve-fiber layer of the retina. The entire fundus, including the blood vessels, had a pale, milky appearance.

In the left eye vision was light perception in the lower field. The ophthalmoscope showed a large retinal detachment over the entire lower half of the fundus. The upper half of the fundus was similar in appearance to that described on the right side. There were many large vitreous opacities. Leukemia was suspected and verified by the blood cytology, as follows:

Laboratory findings (Mr. E. B. Burchell M. Sc.):

White blood cells	512,000
Differential count (difficult to classify):	
Myeloid series over 90 percent and morphologically that of an extremely embryonal type of cell. About 5 percent	

seemed to be differentiating into the eosinophile type.

Red blood cells 2,065,000
Hemoglobin .....46 percent

Physical examination: Negative, except for a large liver and spleen, reaching almost to the pelvic rim.

Fig. 2 (Reese and Guy). Myelogenous leukemia with exophthalmos (right). Fullness and protrusion of upper lid and edema of bulbar conjunctiva.

The patient was transferred to the New York Hospital. On the sixth day after he was first seen, an exophthalmos was noted on the right side (fig. 2). An examination of the interior of the right eye at this time showed an extensive retinal detachment with a large vitreous hemorrhage. The condition of the left eye was the same as at the first examination, which was essentially similar to that now noted in the right eye. On the tenth day a sudden exophthalmos occurred on the left side (fig. 3). This was of an extreme degree, the eyeball protruding beyond the palpebral aperture and literally lying on the cheek. The bulbar conjunctiva was pushed out and exuded sanguineous serum. Because of the exposure of the eye and the resulting discomfort, an enucleation was done. At the time of the operation the orbit was found to be completely filled with blood and a microscopical examination of the globe showed the retinal detachment to be due to choroidal hemorrhage. On the fifteenth day the patient died.

Discussion. First in the sequence of events was a choroidal hemorrhage, with detachment of the retina in the left eye, causing the patient to visit the eye clinic. Six days later an orbital hemorrhage with exophthalmos and a choroidal hemorrhage with detachment of the retina occurred on the right

Fig. 3 (Reese and Guy). Myelogenous leukemia with exophthalmos (left). Protrusion of globe beyond palpebral aperture and marked distention of bulbar conjunctiva with hemorrhage.

side. This was followed, five days later, by an enormous expulsive hemorrhage in the orbit on the left side.

Hemorrhages in any part of the body form a well-known part of the picture of myelogenous leukemia. In addition to a tendency of the blood elements to break through the limits of the blood-vessel walls, there is also an increase in the blood-clotting time. It is not surprising therefore, although exceedingly rare, to encounter in this disease orbital hemorrhage with exophthalmos and choroidal hemorrhage with retinal detachment.

73 East Seventy-first street.

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CHOLESTERIN CRYSTALS IN THE ANTERIOR CHAMBER

DR. JULIUS FEJER
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(TRANSLATED BY DR. W. H. CRISP)

Two cases of this rare condition, one occurring in an eye which was the seat of chronic uveitis and detachment of the retina and the other in an eye with shrunken lens following injury, are described. There was a slight increase above normal in the blood cholesterin in one case but in the other it was normal.

In older persons cholesterin is usually found in the eye in the form of glittering structures in the vitreous. The clinical picture of synchysis scintillans is well known, and is occasionally to be seen in persons who have full visual acuity. Cholesterin crystals also occur now and then in the degenerate, hypermature lens, in the various senile corneal degenerations, in the optic nerve, or even with extreme rarity, in the choroid. Only with great rarity are they encountered in the anterior chamber, in large masses.

My present case is that of a forty-four-year-old man whose right eye was treated in my division seven years ago for retinal detachment with high myopia, by the methods customary at that time. His condition was improved only in slight degree, from counting fingers at 0.5 M. to counting fingers at 1.5 M., the visual fields becoming materially larger, and the retina being reattached, although only over a small area. The improvement lasted only a short time, and the patient then disappeared from my observation. Three months ago he turned up again and complained of moderate pain and complete blindness of the affected eye.

Examination disclosed the following conditions: the iris tissue atrophic, the whole pupillary border adherent to the anterior surface of the lens, cloudy, the peripheral parts of the iris bulging forward—a picture such as we are accustomed to see after degeneration of an eye from chronic uveitis. There was no light perception, tension was 38 mm. Hg. The striking feature was the presence of a strange silvery, naphthalin like, glittering, flaky substance which fluttered here and there in the anterior chamber in such masses that the floor of the chamber and the posterior corneal surface were covered to a height of about 1.5 mm. Examining with the slit-lamp these mobile structures, some of which adhered to the anterior surface of the iris, their delicate outlines were clearly visible. They were octagonal,

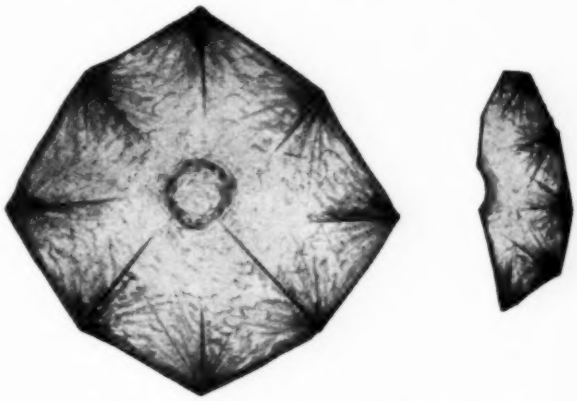

Fig. 1 (Fejer). Front and lateral views of cholesterin crystal.

with linear divisions, and an umbilicate central depression. Figure 1 shows such a tiny crystal in front and side views. Dr. Papolczy was good enough to prepare a stereoscopic picture of the eye which very clearly demonstrates the glittering crystals in the anterior chamber (figure 2).

In this case we have to deal with a chronic uveitis which was also the cause of the retinal detachment and of the degenerative changes as well as of the cholesterin formation. How these crystals reached the anterior chamber cannot be determined exactly, since no communication existed between the posterior chamber, or vitreous, and anterior chamber. Thus we cannot

assume that the cholesterin arose from the vitreous, and it is much more probable that it was secreted from the blood by diapedesis as a product of degeneration. Heilickmann believes that in such cases the consistence of the contents of the anterior chamber suffers some sort of change which leads to precipitation of cholesterin. The cholesterin content of the blood amounted to 174 mg. per 100 c.c. (against a normal of 140 to 170 mg.) The Wassermann and Meinicke reactions were negative, and the urine showed no pathologic constituents.

Only a very few similar cases have been recorded in the literature. Noteworthy is a case of Bartok (1921) in a woman of forty-nine years with traumatic cataract and silvery, silky flakes in the anterior chamber, which reformed after puncture and emptying of the anterior chamber. The substance was identified both chemically and microscopically. Similar cases have been described by Heilickmann, Meesmann, Evans, Derkoe, Greeves, Adrogué, Rohrschneider, Stock, and Alajmo. Meesmann undertook experiments on albino rabbits, injecting a cholesterin suspension into them and enucleating the eyes after eight weeks. He found changes similar to those encountered in his patient and he believes that the cholesterin reached the interior of the eye by becoming dissolved in the aqueous. Many believe that the cause of the cholesterin formation is to be sought in the blood, in a hypercholesterinemia. Fischer-Galatzi found 371 mg. of cholesterin per 100 c.c. in the blood of his patient.

After seeing no similar case in the course of several decades, the proverbial tendency to repetition of cases was exemplified by the recent arrival of a second such clinical picture. This was in a woman of sixty-one years who had injured her right eye ten years previously with a needle, the complications being cataract formation, shrinking and dislocation of the lens, and complete blindness. Similar cholesterin crystals were to be seen not only at the bottom of the anterior chamber, but also distributed in the trabecula of the atrophic iris in the form of fine, glittering granules. The blood cholesterin was 143 mg. per 100 c.c., and was therefore not above normal.

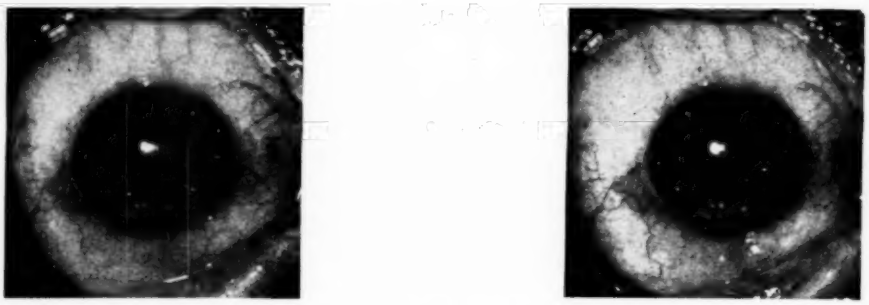

Fig. 2 (Fejer). Stereoscopic illustration, showing crystals.

Inspection of the literature indicates that the formation of cholesterin in the anterior chamber had in most cases been preceded by injury and cataract.

Nador-Utca 11.

SOCIETY PROCEEDINGS

Edited by Dr. H. Rommel Hildreth

COLORADO OPHTHALMOLOGICAL SOCIETY

December 17, 1932

Drs. D. A. Strickler and E. R. Neeper presiding

Episcleral myxofibroma

Drs. D. H. O'Rourke and John Long presented M. M., a forty-two-year-old Mexican, who about 1923, had had a sensation of a foreign body in the right eye and went to a railroad hospital in El Paso because of the eye, but nothing was done. A small red spot, about 1 mm. in diameter had appeared temporalward to the cornea. There had been some pain and slight photophobia associated with this at the time. Since 1923, there had been a slow, progressive increase in size of the mass up to four years ago. In 1928, the mass was large enough to protrude between the lids and since then had caused more pain, particularly while in the wind. Sight had been unaffected. General health had been good.

The mass was now globular, about 2 cm. in length and 1 cm. in width. The long axis was vertical when the lids were separated. It transilluminated readily and was attached firmly at its base. There were numerous dilated blood vessels over its surface. Ophthalmoscopically the eye was negative.

On December 21, 1932, under local anesthesia, the mass was removed. A vertical incision was made in the conjunctiva and the mass separated by sharp dissection. A hypodermic needle was thrust into and through the mass but no fluid could be aspirated. The base of the mass was ultimately associated with the tendon of the external rectus. The muscle was not cut. At the conclusion of the operation the external ocular movements were normal. Heaing was uneventful and on January 28, 1933, there was no evidence of recurrence. The pathological diagnosis was myxofibroma.

Luetic (?) iritis

Dr. C. E. Walker presented Mr. T. W. B., a twenty-nine-year-old fairly healthy but not robust man, who had received a wound on his hand in April, 1932, which would not heal. About the same time he developed a severe pharyngitis, the posterior and side walls being covered with a thick adherent white membrane. The throat was very tender and did not respond to local treatment. A Wassermann reaction was reported three plus, and under intensive antiluetic treatment the throat cleared. In August, iritis developed in the right eye; after it had progressed for two weeks he was seen by Dr. Walker. Another Wassermann reaction was positive.

In spite of atropin, dionin and heat treatment and the removal of four teeth, the process had continued so that now there were present posterior synechiae, circumcorneal redness, increased tension, keratitis punctata, and vision of fingers at two feet. The left eye had a few floaters, but there was 20/20 vision. The record of another oculist showed that the vision of both eyes was normal in 1924.

Discussion. Drs. Melville Black and E. M. Marbourg suggested a subconjunctival injection of atropin and adrenalin to loosen the synechiae. Dr. Black thought the eye might be tuberculous.

Dr. E. R. Neeper said that he would increase the use of mercury in addition to the arsenicals.

Drs. W. H. Crisp and C. E. Sidwell recommended having the patient examined for a gonorrheal focus. Dr. Sidwell said that gonorrheal cases were usually more painful than this had been.

Dr. Wm. C. Bane said that it seemed to him that by this time the throat condition should warrant removal of the tonsils.

Dr. D. H. O'Rourke believed he could see a large exudate in the fundus

which combined with the keratitis punctata would warrant a diagnosis of uveitis rather than iritis.

Dr. G. L. Strader said that his procedure in forcing synechiae loose was to instill three percent cocaine, five percent atropin, and then an adrenalin pack and then to follow with the high frequency current.

Dr. F. R. Spencer advised prostatic massage.

Dr. G. H. Stine said that he had recently done a paracentesis several times on a case of glaucoma secondary to iritis, and that now he could see a white object through the pupil and wondered whether the repeated moderately sudden reductions of tension had not caused a retinal detachment.

Corneal opacities, epithelial dystrophy, and glaucoma following cataract extraction

Dr. George H. Stine reported further notes on the case of Mr. J. O. M., (American Journal of Ophthalmology, April 1932) who had developed opacities in the substantia of the cornea and epithelial dystrophy, following a cataract extraction, which had been done in July, 1931. Cyclodialysis was done in October, 1931, for glaucoma, which appeared early in October. Following the cyclodialysis the tension remained within normal limits until October, 1932. There was however, little change in the corneal dystrophy during this time. In May, 1932, the patient developed a series of small epithelial erosions which he felt were due to straw chaff lodging in the eye. However, the ulcers were evidently due to rupture of the corneal bullae. This condition apparently quieted down for three months, when in August the patient returned complaining of scratching and burning of the eye. At this examination the corneal epithelium was found to be very edematous, with a large bulla in the temporal portion, and two deep, yellow infiltrates in the nasal portion. The tension at this time was 25 mm. of mercury (Schiötz). The corneal ulcer responded to treatment.

Early in October the entire cornea was denuded, except for a peripheral 2 mm. margin all around. The tension was elevated to at least plus two by palpation. There was also a deep disciform infiltrate in the lower nasal portion of the superficial ulcer. The eye was treated locally with heat, atropin, dionin, thermophore, cauterization with iodine and phenol, and foreign protein therapy, without any influence on the progress of the condition. The patient suffered severe pain. After four weeks of such treatment corneoscleral trephining was done. During the operation it was found that the bulbar conjunctiva of the nasal, upper and temporal portions of the globe was firmly bound down to the globe in the limbal region. Owing to this scar tissue and the inelasticity of the conjunctiva, it was impossible to draw conjunctival flaps across the cornea from the nasal and temporal portions of the globe as was intended. Following the operation there was a definite decrease of tension to normal or below, with marked relief of pain. The ulcer responded rapidly to treatment, so that in a month's time it was completely healed. There was a rather dense leucoma occupying the entire central area of the cornea, with numerous new vessels, deep and superficial, running to the area. The epithelium of the entire cornea now seemed quite normal. However, the tension gradually rose without any pain or other symptoms. This was apparently due to the growth of new scar tissue of the conjunctiva and episcleral tissue around the trephine opening as the small, black trephine hole could be seen through the conjunctiva; there was no bleb. The eye had light perception only, and that through the periphery of the cornea.

Dr. Stine reported the case because of the clearing of the corneal epithelium, in spite of the subsequent rise of tension. He felt that this was due to the marked increase in the nutrition of the cornea itself. He asked if it would be worth while to do another trephining on an eye in which the prospects of regaining useful vision were so very poor.

Discussion. Dr. Melville Black suggested doing another trephining, to the side.

Dr. D. H. O'Rourke suggested cyclodialysis because it could be repeated.

Glaucoma cup from arteriosclerosis (?), and delirium secondary to atropin

Dr. C. E. Walker reported the case of a fifty-eight-year-old farmer who had been noticing lowered vision for over a year. About February, 1932, he had gone to another oculist who had found subnormal vision, marked concentrically restricted fields with practically no color vision, quite definite glaucomatous cupping with marked pallor of the discs and with slightly increased tension on only one of many trials. The blood Wassermann test had been negative and urinalysis had been practically normal; B.P. 152/88. A second visual field taken in August showed improvement. The patient had been using pilocarpin.

One night in the middle of September, about forty-five minutes after the first instillation of a refill of the prescription, the patient developed delirium, flushed face, and increased pulse rate. The symptoms lasted about fifteen hours and recurred on the following day when the drops were again used. The general practitioner called, thought that atropin had been used in making up the prescription although the pupils were not dilated. Unfortunately the solution had not been saved.

About October 1, 1932, the patient reported to Dr. Walker with the previous history. The vision was 20/40 O.D. and 20/70 O.S. corrected. The appearance of glaucoma cups was still evident and the tension was elevated, to fingers. By confrontation the fields seemed about normal. Dr. Walker again ordered pilocarpin believing the symptoms had been due to the mistaken use of atropin by the druggist.

Dr. Walker wondered whether the appearance of glaucoma cups was not really due to arteriosclerotic changes in the blood vessels at the base of the brain, thus fitting into the group as recently reported by Knapp, although no such blood vessels could be demonstrated by x-ray in this case.

Discussion. Dr. G. L. Strader said that in thinking of these cases of glaucoma cups without increased tension that perhaps what was called normal tension according to the tonometer reading was really abnormal for that individual, because of a weak lamina cribrosa.

Dr. J. A. McCaw called the attention of the society to the case that he had presented several years previously, as one in which the tension had been held within normal limits for thirteen years by the use of pilocarpin and massage. Dr. McCaw said that recently this individual was losing part of his field and central vision in spite of the fact that the tension was always normal when taken. This patient had lost the first eye from glaucoma.

Dr. G. H. Stine said he had recently seen a patient with a glaucoma cup, a typical Bjerrum scotoma, and normal tension. This patient was given pilocarpin. Dr. Stine wondered whether allergy was not a factor in glaucoma. He doubted whether such cases should be trephined if the tension could never be found elevated.

Dr. E. R. Neeper said that he remembered of having seen about six cases of cupped discs, with tension, fields, and vision normal and that he had termed this interesting appearance of the fundi as "countersunk discs."

R. W. Danielson,
Secretary.

ROYAL SOCIETY OF MEDICINE, LONDON

Section on Ophthalmology

February 10, 1933

Mr. A. C. Hudson, president

Congenital dislocation of the lenses

Mr. Adrian Caddy reported the case of a woman aged twenty-nine years who had been troubled with high myopia and congenital dislocation of the lenses since childhood. With —18.00 D.sph. and—4.00 D. cyl. for each eye her vision was 6/36. Gonin's operation was carried out, but with little success, but in January last, while in bed, she noticed that the vision of the right eye suddenly improved, and that the wear-

ing of her spectacles spoiled her vision. It was discovered that the lens had slipped into the vitreous. With a +4.00 D.sph. she had a vision of 6/9. This was an example of natural couching of the lens.

Congenital hyaline membranes on the posterior surface of the cornea

Miss Ida Mann said the association of hyaline membrane formation at the periphery of the posterior corneal surface with abnormality of the iris stroma and normal intraocular tension appeared to be a rare congenital anomaly. In a young man aged twenty-nine years it involved the left eye only and visual acuity was normal. Tension was also normal, and the condition was observed accidentally. The membrane was applied to the posterior surface of the cornea around most of the circumference. The anterior layer of the iris stroma was composed of abnormally thick strands which overlapped the pupil margin when this was dilated. A second case was in a child aged eight years who showed the condition in both eyes. She had myopic astigmatism and slight lens opacities with poor visual acuity. In both eyes the hyaline membrane was present on the deep surface of the cornea and there was abnormality of the iris stroma.

No record of an entirely similar case had been found, although Stephenson, Coats and Herbert had discussed the question of the formation of hyaline material on the cornea; and Ballantyne had shown a case resembling the present one in some respects. The suggestion was made that the condition was a pure developmental aberration arising from abnormality of the post-endothelial tissues of the embryo.

Safar's method for treatment of detachment of the retina by diathermy

Mr. J. Cole Marshall reported the following case: A man aged fifty-eight years, with photopsia, which had been noticed one week, in whom "everything appearing smudgy, then all was darkness," was able later to see daylight. A series of 14 diathermic punctures was made in an oval form, and included a series of tears. Extra punctures were made under the tendon of the superior rectus. The patient's field of vision was now full.

Safar, said Mr. Marshall, aimed at getting discrete spots of coagulation in the choroidal layer in such a way that no injury occurred to the retina, nor destruction or weakening of the sclerotic. In comparison with Larson's method it seemed more exact in every way, as one could be sure of only a very small effect on the sclerotic, and no serious damage beyond the point of puncture, especially in cases with tears near the macula or the ora serrata. This was due not only to the needles employed, but also to the smallness of the current needed. By using a special series of nails it was possible to make large barrages without the eye becoming soft. The technique was almost the same as in the Guist method of encircling or creating a barrage. Safar's method avoided the need for resecting muscles. Safar now used a one-pointed needle for "strippling," which he did in large disinsertion areas, but he did not do this until he had carried out his barrage and removed the nails, so that subretinal fluid had been let out, and the retina between the edge of the tear and the ora was lying flat.

The nails were held by the operator in a special pair of isolated forceps, and when the needles were applied against the sclerotic the forceps were touched by the pole, and the current at once enabled the nails to be inserted into the sclerotic. When the three points were well in place, the forceps were opened and the nails left in situ. If punctures were made over the area of healthy retinal tissue, secondary tears were not likely to occur, as the scars were away from the margin.

It was a great help to have specially made retractors with ivory heads, also flat ivory retractors, bent at a slight angle.

With regard to the evacuation of the fluid, if enough fluid did not appear to have been evacuated when the needle was withdrawn, or the nails had been taken out, further fluid could be reremoved by putting the blunt fine noz-

zle of an Anel's syringe in the puncture holes and drawing off the subretinal fluid.

The patient, as soon as the diagnosis had been made, should wear Lochbrille spectacles, and be kept in bed for a week before the operation. After the operation both eyes should be bandaged for fourteen days. The first dressing was done on the fourth day and the fundus was quickly examined. At this period it was important to prevent the patient trying to use the eye. It was desirable to keep the patient in bed until the end of the third week, wearing Lochbrille spectacles. After Safar's operation the results were not seen so soon as after Gonin's, as they might only definitely appear at the end of the first week. The condition of the retina might continue to improve until the fourth week. No second intervention was carried out until at least a month after the first operation.

Areas were seen, about the size of the optic disc, round, at first of a pinkish-yellow, and stippled with black pigment. Gradually the pigment became more marked and discrete, and the background whiter. Retinal vessels were seen lying flat over the scar. The vitreous cleared rapidly, and no vitreous hemorrhages had occurred in his cases.

With regard to results achieved by Safar, up to 1932, he had carried out the operation in 39 unselected cases, and among them he had 24 reattachments, while in recent cases of myopia and after contusion more than 90 percent.

(Reported by H. Dickinson).

CHICAGO OPHTHALMOLOGICAL SOCIETY

Dr. Michael Goldenburg, president

January 16, 1933

Dr. Harry Friedenwald was the guest of honor at this meeting.

Retinal vascular disease

Dr. Harry Friedenwald described the groups into which he and Dr. Jonas Friedenwald had been led by long continued clinical studies to divide the cases examined on the medical wards.

1. Hypertension without retinal arteriosclerosis in cases of essential hypertension, acute nephritis, mild toxemias of pregnancy, hyperthyroidism with hypertension, etc. Eyeground normal; slight arterio-venous constriction.

2. Retinal arteriolosclerosis and albuminuric retinitis presenting localized variations in caliber, irregular tortuosity, and visibility of the walls of the terminal arterioles. The larger retinal arteries in early cases were full and tortuous, and often showed a coppery appearance. Arterio-venous constriction was usually present at the points of crossing of both large and small branches. In late stages of this condition the larger arteries also showed marked lesions such as beading, increased visibility of their walls, and even diffuse constriction of caliber. In these cases, both with and without albuminuric retinitis, there was extremely high hypertension, sooner or later associated with a rapidly progressive chronic nephritis.

3. Retinal arteriosclerosis with hypertension in elderly persons with arteriosclerosis: reduction in caliber of the whole arterial tree. Arteries generally straight and branch to acute angles. Blood pressure could usually be estimated from this sign alone.

4. Retinal arteriosclerosis without hypertension resembling that just described, but with this difference, the main arterial branches were full and tortuous. Arterio-venous constriction, caliber variations, visibility of the vessel walls might be present. Elderly persons presenting this ophthalmoscopic picture had, in general, shown no hypertension.

In studying the retinal arterial system, we must bear in mind that we were dealing with "arteries" and "arterioles"; the branches of the first and second order were arteries, the finer divisions were arterioles, but even the largest branches resembled the arterioles in the thinness of their walls, the absence or slight development of the muscular coat. The distinction between arteries and arterioles was emphasized by the marked differences in their clinical manifestations. Localized caliber varia-

tions in the larger arteries were usually due to atheroma.

The central retinal artery within the optic nerve was specially liable to this form of sclerotic change.

General attenuation of the retinal arteries appeared to be due to atheromatous changes in the vessel behind the disc with consequent decrescent change, a shrinking in caliber and length of the retinal vessels.

Concerning the light streak, "an increase in the thickness of the media will cause the width of the reflex to increase relative to the diameter of the lumen, while an increase in the refractive index of the media will result in an increase in brightness of the reflex." This explained the copper-wire arteries found in cases of arteriolosclerosis. It was only when the arterioles with hyaline thickening were infiltrated with lipoid substance that they appeared as "silver wires".

Arterio-venous constriction was produced by a number of factors: at points of arterio-venous crossing the adventitia of the two vessels formed a continuous fibrous coat, so intimate that it became impossible to say where the arterial wall ended and the vein began. Thickening of the arterial wall would reduce the caliber of the vein. The sclerotic patch in the arterial wall sometimes penetrated the vein causing an actual phlebosclerosis. An actual displacement of the artery due to changes in tortuosity or in length would drag the vein out of its course.

There was mention of fullness and tortuosity of the larger retinal branches only in retinal arteriolosclerosis. Full arteries were generally the evidence of an absence of hypertension. But the exception just mentioned demanded great care in examining for signs of arteriolosclerosis of the macular arterioles especially in the earliest stage.

There was little evidence of primary sclerotic venous changes. White streaks seen along the borders of the veins were due to wandering cells in the perivascular spaces, the "foam cells" carrying off the detritus from retinal degeneration. Doubt was expressed concerning Guist's recent observations that all the veins were diseased, and that their walls presented hyaline changes, and could with difficulty be distinguished histologically from arteries.

There had been much study during many years concerning the part played by the arterioles in the production of albuminuric retinitis. The lecturer presented the evidence recently furnished by Dr. Jonas Friedenwald showing that the arteriolar lesions were " . . . scattered throughout the retina, and that they were definitely related to the focal lesions, hemorrhages, and so-called exudates characteristic of albuminuric retinitis".

The outstanding facts that the study of the retinal vessels had brought out were:

1. The sharp distinction between arteriosclerosis and arteriolosclerosis. They differed in their ophthalmoscopic appearance, in their local sequelae, and in the groups of general disease in which they occurred.

2. Retinal arteriosclerosis gave indication of the degree to which general arteriosclerosis had developed, and furnished a measure of what the arterial tension would otherwise be in conditions when this was depressed by cardiac decompensation.

3. The retinal vessels might show changes in spite of hypertension of long standing. But when arteriolosclerotic changes in the retina appeared there was always hypertension with arteriolosclerosis already present or likely to develop in the kidneys. Thus it might happen that the eyegrounds showed marked changes, without functional disturbance of the kidneys, or vice versa. But generally in these conditions both sets of organs were affected.

Robert von der Heydt.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

Section on Ophthalmology

October 20, 1932

Dr. L. F. Appleman, chairman

Unilateral pulsating exophthalmos

Dr. L. F. Appleman presented a male patient, first seen on October 5, 1932.

Ten days previously the man had been struck with a fist on the right eye. On the morning following the injury there was a soreness over the right side of the head and diplopia was noticed on the third day. Examination showed a complete oculomotor and abducens nerve paralysis. The globe pulsated and a bruit was heard over the temporal region. The retinal veins were very tortuous, otherwise the eye was normal. The spinal fluid was negative to the Wassermann test but contained red blood cells. No fractures were revealed by x-ray. Probably a ligation of the carotid artery, or branches, would be required in treatment of the condition.

Amaurosis after uterine hemorrhage with restoration of vision following transfusion

Dr. H. Maxwell Langdon and Dr. George M. Laws presented a patient aged thirty-nine years, who was admitted to the Presbyterian Hospital March 14, 1932. The history was that for two years there had been attacks of recurrent vaginal hemorrhages, often very profuse, lasting from three to fourteen days. The last attack started fifteen days before admission.

Vaginal examination revealed a large fibroma of the cervix; hemaglobin 45 percent, red blood cells 1,800,000 and white blood cells 22,000. The day after admission she was given 300 cc. of citrated blood intravenously, with improvement in her general condition. The third day after admission the patient became drowsy and somewhat delirious. Another transfusion of 500 cc. of citrated blood and 500 cc. of 20 percent glucose was given with some slight improvement. On the fourth day another transfusion of 500 cc. of citrated blood was given.

At this time the patient complained of blurred vision and an ocular examination was made; the pupils were sluggish to light, the media were clear, the discs were covered with a white fluffy material, the margins being completely obscured. There were no intraocular hemorrhages. The following day her general condition had improved, the blood count having risen to 2,719,000 and she could count fingers at two feet. The general condition continued to improve but on the ninth day, vision was reduced to questionable light perception and there were no pupillary responses to light. The fundi were generally pale and the discs less edematous. Two weeks after admission the vision was 1/3 of normal and the discs much less edematous and of better color. The pupils responded well to light and there were no ocular palsies.

A hysterectomy and appendecomy were performed, the patient made an uneventful recovery and four weeks later the vision was O.D. 6/12, O.S. 6/6. The form fields were constricted to 40 degrees. Four months after discharge from the hospital the vision was O.D. 6/7.5, O.S. 6/6. The discs were slightly pale, but otherwise the fundi were negative. The patient's main complaint was "things are dark" and with the disturbance of the retinal cell elements which Ward Holden had shown to occur in this type of case, it seemed very probable that there had been a disturbance of her light sense; unfortunately the patient's mental condition made these tests very unsatisfactory.

Conditions similar to those in this case were a well recognized medical entity and usually ended in serious loss of vision if not blindness. The loss of vision following anemia from loss of blood seldom, if ever, occurred after a traumatic loss of blood but almost invariably in association with diseased conditions. It might occur after prolonged hemorrhage as in this case or after a sudden much larger loss of blood. It had been reported after hemorrhage from the lung, from the gastro-intestinal as well as the genito-urinary tracts. Before transfusions became a standard treatment for such acute anemias the prognosis for vision in these cases was very bad as the affected retinal and cerebral cells went on to degeneration with ascending atrophy of the optic nerve before the blood condition could be brought to a stage which furnished them sufficient nutriment, the treatment, of course, being much more prolonged and slower in its results. It was not likely that ophthalmologists

would be the first to see such a case but it should be borne in mind by any physician irrespective of his special interest in medicine, that massive transfusions offered a method of preservation of vision which could not be duplicated in any other way and should be resorted to at once. The improvement in vision in the patient here recorded, after large transfusions were used, was so prompt and extreme that it would seem that a good prognosis could be given in any case if transfusions were made use of promptly. It would also seem that the anemia was the sole factor in the production of the neuro-retinal condition and where this was overcome, no other factor remained to be considered.

Discussion. Dr. Maxwell Herman said that he had had a case of similar nature which followed a post-partum hemorrhage in a normal obstetrical case, with the simple history of the patient complaining of the sudden loss of sight. It was the typical picture such as a case described by Dr. de Schweinitz which was followed by optic neuritis, swelling of the nerve head, marked pallor, and typical macular pallor with cherry red spot. After seventeen days she was re-examined and it was found that she had vision enough to read ordinary print.

Dr. Maxwell Langdon said that there was a generalized fundus pallor with veiling of the disc and macular region but no accentuation of the pallor over the central portion, nor evidence of the cherry red spot at any time.

Dr. George M. Laws in reply to Dr. Shumway said that repeated blood examinations showed that the anemia was a purely secondary type. The appendix was taken out, partly as a routine measure and partly because the patient had had some vague pain in its region. The laboratory report was "chronic appendicitis" but there was no evidence that it was capable of causing toxemia.

Dr. Burton Alexander Randall as an ophthalmologist

Dr. Burton Chance read a paper on this subject which was published in this Journal, v. 16, no. 6, p. 504.

A. G. Fewell,
Secretary.

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MILTON'S BLINDNESS

Many, but not most, of the readers of this Journal will have read the essay recently published by W. H. Wilmer on "The blindness of Milton" (Bulletin of the Institute of the History of Medicine —Supplement to the Bulletin of the Johns Hopkins Hospital—1933, volume 1, April, pages 85-106).

The controversy as to the cause of blindness in the author of "Paradise Lost" and "Paradise Regained" has occupied his biographers, his literary critics, and the ophthalmologists. Thus, in 1879, the view that it was due to glaucoma was advanced by his German biographer, Alfred Stern. The same diagnosis is suggested by Hirschberg in a footnote to his "History of Ophthalmology," and it was repeated by Thomas H. Shastid in an article on Milton in the "American Encyclopedia of Ophthalmology." According to Mutschmann, Hirschberg's opinion was based upon Milton's own statement in a letter to his friend the Greek diplomat Philaras that "if he looked at a lit candle a kind of iris seemed to snatch it" from him.

The nature of Milton's blindness was very ably and interestingly discussed by Arnold Sorsby in the British Journal of Ophthalmology three years ago (1930, volume 14, page 339). There is always something inspirational and appealing about the blind man who preserves his moral courage to the point of doing distinguished work in spite of his affliction. Sorsby's paper was fittingly introduced with the following autobiographical allusion from Milton's "Paradise Lost":

". . . Thus with the year
Seasons return, but not to me returns
Day, or the sweet approach of even or morn,
Or sight of vernal bloom, or summer's rose,
Or flocks, or herds, or human face divine:
But cloud instead, and ever-during dark
Surrounds me, from the cheerful way of men
Cut off, and for the book of knowledge fair
Presented with a universal blank
Of Nature's works to me expunged and rased,
And wisdom at one entrance quite shut out."

The development of Milton's blindness appears to have begun in 1644, when he was about thirty-five years old,

and to have been complete by 1652, when he had attained the age of forty-three years. In addition to less direct references, Milton gave a fairly detailed account of his own case in the letter to his friend Philaras. Here he used the sentence above quoted, presumably describing a halo around the candle light. His sight had grown gradually "weak and dull," his eyes had been "at once thoroughly pained" if he attempted to read. Darkness had "come over the left part of my left eye" (that eye being "clouded some years before the other"). The other eye had failed "perceptibly and gradually through a period of three years." Yet, as mentioned on several occasions by himself and others, but especially in a sonnet dealing with his blindness and addressed to his friend Cyriack Skinner, the eyes were "clear to outward view, of blemish or of spot."

Wilmer favors strongly the conclusion that Milton was a sufferer from simple glaucoma. He bases this belief upon the symptoms above selected for quotation from the letter to Philaras, and on some details available from other sources; particularly emphasizing the gradual failure of vision in the left eye, followed later by a similar condition in the right, the temporal and upper visual field defect in the left eye, retention of a small spot of peripheral vision in the right eye after loss of central vision and after complete blindness had developed in the left eye, and the halo around the candle flame.

But Sorsby, while admitting that the general outline of the case gives color to the diagnosis of glaucoma, objects that a number of details speak against this conclusion. The evidence, he says, "presupposes the glaucomatous process to have started very early in life if by the early thirties the sight of an eye could have been lost. . . . It is surely most unusual for chronic glaucoma . . . to lead to complete blindness by 43. . . . Chronic glaucoma is essentially a symptomless disease. . . . Glaucomatous patients do not describe loss of field, such as Milton describes. The metamorphopsia of which Milton speaks, the micropsia, the illusion of objects moving, the 'copious light glittering from shut eyes' are all symptoms that are never met with in glaucoma. . . . When Milton was blind, he could still get 'a certain little trifle of light.' . . . The blindness from glaucoma is usually absolute."

Sorsby apparently leans toward the assumption that Milton became blind from retinal detachment secondary to myopia, as argued by Dufour and others. The grounds suggested for such diagnosis include the "illusions of deformity and mobility of objects looked at, the subjective sensations of light persisting after loss of sight, and . . . 'dimness of the visual field coming on first on the left side and then at the top; the sensation of steam before the eyes.'"

Dealing as it does with one of the giants of English literature, the discussion has a bearing upon our sense of the havoc wrought by such diseases as glaucoma, retinal detachment, and the less frequently favored diagnoses of Milton's affliction.

It is not even established that Milton was highly myopic, although the evidence does suggest that he was myopic to an appreciable degree. The poet's obvious inaccuracies in reference to natural history may be merely those of a city dweller or even the carelessness of so-called "poetic license." As with most lay descriptions of the symptoms of disease, especially when one has to rely upon distorted memories extending over a long period of years, the patient's account of his own history is none too reliable, and does not fit in exactly with any single clinical picture as known to modern ophthalmologists.

Sorsby's objection that "it is most unusual for chronic glaucoma . . . to lead to complete blindness by 43" is ineffectual. It is most assuredly possible for simple glaucoma to produce complete blindness before the age of forty-three years, and Milton may have been just as exceptional in this as in his poetic genius. The writer of the present comment has a patient whose simple glaucoma in both eyes began at least by the age of twenty-nine years, and whose field encroachments extended almost to the centers at the age of thirty-eight years, eight years after bilateral iridectomy by her previous surgeon in an-

other city, with vision of the worse eye reduced to about 3/30.

Neither the diagnosis of glaucoma nor that of retinal detachment is thoroughly established in Milton's case. But Wilmer's preference for simple glaucoma appears to find substantial support in the evidence furnished by the patient and his contemporaries. In a personal communication, Dr. Wilmer emphasizes "the flimsiness of the opinion that Milton's poor observation of Nature was due to nearsight." "In fact," he says, "Milton was an unscientific city man, who enjoyed country sights and sounds without knowing anything about them. . . . In 'L'Allegro' he has confused the robin and the lark. . . . He also distinguishes . . . between the sweet-briar and the eglantine, though they are the same plant. . . . He also speaks in the 'Arcades' of the foliage of the elm as if it were extremely thick. . . . Though the nightingale is vocal for only about a month each year, he writes in 'Paradise Regained' 'The nightingale thrills her thick-warbled notes the summer long.'"

In a sense the debate over Milton's blindness may strike us as futile. But the struggle against natural infirmity awakens a sympathetic cord in us all, and against the gray background of his blindness the magnificent colors of Milton's poetic genius shine forth even more vividly. We experience in regard to him somewhat the same emotion as is inspired by the deafness of that greatest of musical composers, Beethoven.

W. H. Crisp.

BLINDNESS IN PALESTINE

According to The Lancet annotations, of April 29, 1933, Palestine has the highest percentage of blindness of any civilized country of the world. This is shown by a careful census taken in 1931. For each hundred thousand of the population 843 were totally blind in both eyes. This is to be compared with 776 in Egypt, 420 in Cypress and only 73 in England. The figures for the United States would be about the same, or a little less, than in England.

The test of such blindness was inability to count fingers at one meter distance. This is about one half as much vision as is counted industrial blindness in America. Of the blind applying at St. John Ophthalmic Hospital in Jerusalem the blindness in 80 percent could be traced to conjunctivitis, and in a special survey of eleven villages, out of 1,118 blind eyes, 792 were found due to this same cause. "Almost every peasant in the country is infected with trachoma." The incidence was less among the towns-people, but 90 percent of patients of the above hospital were suffering from it.

Among 3,408 infants infected with acute conjunctivitis, about 7 percent of the eyes were hopelessly lost, and as many more had scars from corneal ulcers. Among over 3,000 patients suffering from trichiasis, 393 were operated upon for its relief. The Government has undertaken to organize hospitals and traveling clinics, on somewhat the same plan as has proved efficient in Egypt. It is also proposed to organize a school, where native girls can be trained, to go into villages and teach the elementary rules of hygiene, sanitation, first aid and home nursing.

Edward Jackson.

TRAUMATISM IN RETINAL DETACHMENT

In the proceedings of the Fourteenth International Congress amongst the discussions on detachment of the retina one finds a new interest manifested in the role of traumatism as an etiological factor. In the tremendous assemblage of facts by Arruga upon the etiology and pathogenesis of detachment of the retina there is one section devoted to the influence of traumatism. Analysis of this yields the following facts.

Detachment of the retina immediately following traumatism is rare, exemplified by the observation that in the war injuries few such cases were reported, although rupture of the choroid was frequent. Contusions completely disorganizing the eye may produce immediate detachment of the retina, but these seldom come into medico-legal disputes because of the existent retinal detachment. Laceration at the macula,

however, a not infrequent injury, may produce immediate detachment of the retina and comes in this group.

The cases which are most frequently the source of medico-legal complications are included under the second heading of Arruga; injuries with secondary production of detachment of the retina. In these cases the detachment occurs within from several days to several weeks after the injury. The contusion may have been directly to the globe; although more frequently to the head or periorbital region. At first the patient notices nothing subjectively, but later observes transitory opacities and photopsias. Finally detachment of the retina and obscuration of vision occur, for which the patient consults his oculist. Formerly these slight blows and contusions which were mentioned in the history, or overlooked entirely, were often discounted as etiological factors. Recent study of the pathogenesis of detachment has indicated that the tear is an important factor, so the history of trauma, no matter how slight, has assumed greater significance.

Concerning this type of case Jeandelize and Daudot feel that the history of injury is of the greatest medico-legal importance. They conclude that in detachment of the retina of an apparent spontaneous type, trauma is of frequent occurrence, even though it be of a minimal nature.

Davidson calls attention to Gonin's study of forty-five cases of detachment of the retina, to determine whether they are compensable or not. The lesions produced in the retina were identical, whether caused by direct or indirect injury. Where indirect injury caused the detachment it usually became visible in a period of less than two weeks after the injury. The patients of this group tended to be senile or myopic. Where the detachment was caused by direct injury the interval between injury and detachment was usually longer than three weeks. Upon the basis of his study Davidson suggests that industrial cases of detachment of the retina are entitled to compensation if the existence of an accident can be established, if the patient complains of blurred vision within two days of the accident, and if the detachment occurs within two weeks of the accident.

The third group considered by Arruga lists cases of traumatism with late production of detachment of the retina. This includes chiefly late detachment following cataract operation and penetrating wounds of the globe where the cause is contraction of vitreous bands and scar tissue. It was concluded that detachment of the retina was most likely to happen following cataract operation in myopic eyes where loss of vitreous occurred.

Detachments following indirect trauma are frequent, and this type of case proves the most troublesome from a medico-legal standpoint. From the trend of thought manifested by the participants in the discussion on the role of trauma in the production of retinal detachment one would conclude that there was a tendency to place the immediate blame for detachment upon what appear to be trivial injuries. Most of these cases were heretofore considered under the group of spontaneous detachment. It is acknowledged that a predisposing cause is almost invariably present, such as myopia, areas of chorio-retinitis, or senile degeneration of the retina. Tears of the retina are most likely to occur in the presence of these lesions, and whether they are the cause of the detachment or not, they render it persistent.

From a survey of the various opinions on the subject, Davidson's suggestion that all cases be considered compensable in which an injury was proved, the complaint of subjective signs of retinal damage made within two days, and a detachment of the retina demonstrated within two weeks of the injury, would appear to be most reasonable. It is to be hoped that definite statements concerning their observations on the role of indirect trauma in the causation of retinal detachment will appear in the future writings of those most interested in this subject so that the courts and insurance carriers will have exact data upon which to base their decisions in these cases.

M. F. Weymann.

OPHTHALMOLOGISTS AND LAY REFRACTIONISTS

Reduced to simplest terms the fundamental aims and obligations of physicians are the prevention and treatment of disease. The ophthalmologist's share is to carry this out, in so far as the eyes are concerned. Everything that pertains to this is a part of his responsibility, whether it be education of the public, instigation of suitable legislation, or medication.

Ever since the discovery that lenses were aids to vision and to ocular and general comfort, glasses have been prescribed. Testing has been done by almost any one who desired to do it from the individual himself, selecting glasses from the itinerant pedler or from the counter of the dime store, to the ophthalmologist highly trained in the arts of refraction. A large percentage of this testing has always been done by others than eye physicians, but it is only within recent years that a serious effort has been made by lay refractionists to organize associations. They have now accomplished this and have succeeded in having laws passed in most states of this country limiting and controlling the testing of eyes. Not being inhibited by medical ethics the members of these organizations have brought their vocation, by extensive advertising, often extravagant and misleading, to the attention of laymen and have become important factors in influencing public opinion with regard to the eyes. There is nothing to indicate that such refractionists will not continue to perform their functions in the future as fully as in the past because of the power of numbers and the backing of important manufacturers and wholesalers for whom they serve as outlets for their products.

In addition to those who have entered this field of work merely as a means of livelihood, there are undeniably able men who are endeavoring to raise the standard of optometry. Ophthalmologists cannot overlook the fact that some of the higher institutions of learning have placed optometry in their curricula and that in such universities very complete courses in optics and refraction are given. If this is doubted by any let them study this department in the catalogue recently published by Columbia University in New York.

There is a fundamental difference between ophthalmologists and others who perform refraction, in that most of the latter are actuated largely by commercialism. By heritage and tradition ophthalmology has drawn its ranks from the best that the nations have had to offer. Theirs has been a comparatively small group, under careful scrutiny, which has maintained a very high standard. A comparison with the other, larger group, heterogenous and, until recently, entirely unrestricted, places the latter at a great disadvantage. The physician's professional education has made him supremely conscious of the need of mankind for help and has instilled in him pity and consideration for those who come to him for medical advice. This is a very important factor in his relation with his patients throughout his life. Nothing in the training of the lay refractionist is comparable to this. His service, for this reason and because he charges only when he sells some tangible thing, incontestably tends to be commercial.

The public would be best served if all refraction were in the hands of well trained ophthalmologists but this is obviously impracticable, for if enough physicians to handle the refraction of the total population needing ocular care, became ophthalmologists, this would mean that more than nine-tenths of the work of each, if evenly distributed, would be devoted to refraction. It is unbelievable that a sufficient number of doctors would desire to enter the ophthalmic specialty with such an outlook. That a medical opinion should accompany any study of the optical system of the eye with a view to prescribing glasses or before exercises are ordered for ocular muscles while the health of the patient may be of vital concern, is surely desirable but it is doubtful if those who believe that all refraction should be performed by ophthalmologists only, have any practical solution to the question.

Obviously we must accept the continued existence of the lay refractionist doing an important percentage of eye testing. Since this is to be a permanent condition and since both the ophthalmologist and the nonmedical refractionist are influential factors in the ocular welfare of the population, despite certain fundamental differences in points of view, it would seem that there should be some contact between the two. This might be done by a committee that would be organized, not for the purpose of discussion of controversial points, but on the contrary with a definite determination to avoid such matters and with the intention of discussing optical matters which have to do with the general welfare.

A number of years ago a committee was formed with representatives from each of the three national ophthalmological societies for this particular purpose. Such severe opposition to any contacts with lay refractionists developed within the committee that the matter was dropped. The committee was continued as a committee on optics and visual physiology and has done valuable service along these lines but the purpose for which the committee was originally formed was a sound one and in spite of all the excellent arguments against conferring with the nonmedical refractionists, the common good can be best served by an interchange of ideas between the two bodies.

Lawrence T. Post.

BOOK NOTICES

Binocular vision and the modern treatment of squint. By Margaret Dobson, M.D. 107 pages. Oxford University Press, London. 1933.

The first four chapters of 44 pages are devoted to the fundamentals of the subject of binocular vision and are well written. The fifth chapter on "The refractor" contains a description of a special instrument with illustrations of it. Minute descriptions always make difficult reading and data derived from the use of an instrument is correspondingly hard to follow. Chapter six describes dynamic retinoscopy in cases of squint and is of considerable interest. Thirty-four pages are devoted to orthoptic training considered from the point of view of eleven different instruments which are on the market. That there are so many instruments for this purpose is very confusing to the ophthalmologist. To point out the merits of each leaves the reader still in doubt as to which is best. Obviously he cannot purchase all of them.

The final four chapters outline methods of treatment. It is hard to evaluate this volume accurately. Throughout the book are valuable suggestions, but there is lacking a systematic unfolding of the subject which would give the reader a clear idea of the procedure applicable in his office for the development of binocular depth perception in the eyes of the squinting child. Much information is given and the subject is quite thoroughly discussed. The reviewer confesses that when he received the book he opened it with great hopes that he would find therein a definite description of a satisfactory method of procedure in these trying cases but the very multiplicity of possibilities outlined has made it difficult to decide which the author advocates.

Lawrence T. Post.

Life-giving light. By Charles Sheard. Ph.D., Sc.D. Professor and Director of Biophysical Research; The Mayo Foundation, University of Minnesota and the Mayo Clinic. 174 pages. Baltimore. The Williams and Wilkins Company in co-operation with the Century of Progress Exposition. 1933. Price $1.00.

This is a book for laymen in which the nature of light and its methods of action in nature are described in terms that are scientific but are readily comprehensible. Though there is very little in the book that would be new to the ophthalmologist the reading is entertaining. It is a volume that can be recommended as of value to those who are interested in the elementary study of light.

Lawrence T. Post.

CORRESPONDENCE

The regulation of the specialties in medical practice

The desirability of some sort of standard for the constitution of the specialist in medicine may be conceded, but that involves a consideration of the encouragements for such a disposition, as well as the minimum qualifications therefor, and Dr. Bierring, President Elect of the American Medical Association, at the recent conference in Milwaukee, voiced a self evident truth when he declared that the making of diplomats, which he advocated, was not a measure for legal enactment. The 55 years of struggle, still going on, to secure adequate legislation for the regulation of practice in general could not be forgotten. Legal control was thus, at best, in the dim-distant future; and it also made possible the recognition of limited cults, like optometry. How Dr. Bierring can hope to make progress through the American Medical Association, as he said, and despite the showing of the work done by the Federation of State Licensing Boards, is something of a mystery. The American Medical Association, since 1904 has been committed to the concept that Organized Medicine must be constituted by the enrollment, through the county and state societies, of every licensed practitioner of regular medicine; and in the main—barring the few nonconformists, it is so constituted. The result, however, has been (as predicted by a few) a hybrid; one part devoted to scientific pursuits, the other to economic policies—a sort of unionization, but without the material penalties for infraction of the rules of conduct adopted by the crafts. And to this absence of material penalties may be attributed a large disposition on the part of the membership to "go as you please," so long as it is within the law relating to licensure.

While the crafts may not be as high-minded as the medical profession it has to be admitted that their form of organization insures united action on the will of the majority; and, for the "general welfare" so much talked about, one may almost regret that the latter did not follow the known workable pattern.

Diplomates will thus continue to be volunteers; recruited from one or the other of two classes: namely, those who would have a desire for close contacts with the "higher-ups"; and those who would see therein an appeal to the public. The *license* must be conspicuously displayed in the office; so, why not the diplomas, or certificates, that have been acquired?

In the new order of things, predicted when the "depression" has worn itself out, let us hope that registration for practice, like registration for voting (including the sworn statement of eligibility) will be all-sufficient; for, in this new order, it is not likely that the rule of "caveat emptor" will be abrogated. In brief, we see again the truth in the old refrain that "half-way doin's is no good for dis worl' or de nex'."

And not a little depends on the encouragements for a life devoted to work within narrow lines. These, in the main, have three origins, to wit: A fancied talent for one particular line; a conclusion, from experience in a class A school, that proficiency in all branches of the art is impossible for any but the super-man; and the lure of larger fees for the time and effort expended.

Despite the fact that history records many instances of men reaching distinction in lines diverse from those first selected, every man will (must) insist on proving, by experience, the truth or falsity of first impressions. The fancied talent is thus on the list of the incorrigibles.

The conclusion that only the superman can be proficient in all branches is logical under the conditions observed; and the schools are largely responsible for it. The curricula provide no lines of demarcation between the research and clinical phases; and the double objective is emphasized so long as therapeutics fills a minor role. If the schools would abandon the idea that their function was to turn out a finished product (and it can't be done in the five years prescribed, or make it ten); would realize that their mission was to provide a foundation adequate for a beginner, plus drill in the use of it; and would stress

the old time virtues of self-improvement, unselfishness, and modesty, some of our problems would be eliminated.

The lure of the larger fees (and quicker distinction, perhaps) will doubtless be with us for a long time. In our years of prosperity many luxuries have been advanced to the necessity class: Social standing, to a degree, depends on the kind of establishment maintained; and an up-to-date office—one that will attract patients, means a considerable outlay. In bounds, it's a laudable ambition.

We have tried medical legislation, and seen the cry of "medical trust" translated to legal recognition of the cults. We have fostered a campaign for the medical education of the public, and heard it dubbed "an advertizing contest between the regulars and the cults —with the cults one jump ahead." We have approved additional titles (F.A.C.P. and F.A.C.S.), and had unfavorable comparisons drawn with those employed by our Anglo-Saxon neighbors over seas. Now, let us sit back, and leave something to evolution. Water will continue to seek its level.

H. B. Young,
Burlington, Iowa.

Simultaneous comparison in subjective testing

499 Beacon Street
Boston, Mass.,
June 30, 1933.

EDITOR, American Journal of Ophthalmology,
St. Louis, Missouri.

Dear Sir:

In my article in the current issue of the Journal (June 1933) "Simultaneous comparison in subjective testing," there is a statement regarding locating the principal meridians which may be confusing to some and I will request you to allow me a little space in order to briefly amplify this point.

Where the approximate direction of the clearest line on some form of line chart has been determined this direction corresponds to the most defective meridian either real or artificial. In hyperopia after fogging, the clearest lines run along the *artificially* made more defective meridian, i.e. the more myopic meridian. The cylinder axis to equalize the lines by *clearing* up the blurred ones must always be placed at right angles to the clearest lines, and therefore along the direction of the blurred lines. Where a plus cylinder is used after *fogging*, the cylinder axis is placed along the *clear lines* and equalizes by blurring these to the same extent.

Since it is generally better to equalize by *clearing* up the lines, the location of the cylinder axis by the cross chart is better made by paying attention to the axis mark at *right angles* to the clearest line from the very beginning. Thus if the clearest lines are approximately vertical, the cylinder axis is considered 180 degrees. The cross chart is placed with the axis mark at 180 and the two limbs on either side of this axis mark, now pointing to 45 and 135 degrees, are compared. If the 45 degree limb is blacker, the cross is turned and the axis-mark moved in the direction of the *other* limb, that is, the axis mark moves from 180 to, say, 170 degrees. If the 135 degree limb is blacker the axis mark is shifted from 180 degrees in the direction of the other limb to, say, 10 degrees and the lines compared.

Suppose the limbs appear alike when the axis is at 170 degrees. The cross is turned so that its limbs point to 80 and 170 degrees. The 80 degree lines will now appear blacker. In myopia or in hyperopia under fog, a minus cylinder axis 170 is used (a plus cylinder axis 170 in hyperopia not under fog) to clear up the 170 degree lines and equalize the two limbs of the cross.

The sentence in the article, "the right axis lies nearer to the direction of the clearer limb," should have stated rather that "the more defective principal meridian, real or artificial, lies nearer to the direction of the clearer limb." This is really the fundamental fact behind the procedure and locating this meridian by the method outlined seems most natural. However, in general work and for a more mechanized routine it seems best to immediately take note of the approximate cylinder axis indicated, which is the direction at right angles to the clear-

est lines, and to work from this position. Place one axis mark in this position and compare the two limbs to either side of it. If one limb appears blacker the cross is turned so that this axis mark moves in the direction of the *other* limb until the two limbs appear alike. When the cross is now set to point to the principal meridians, the lines along the axis will be the blurred lines, which have to be cleared with a cylinder, its axis running in this direction. The numbering on the dial used in the illustration is from 0 to 180 clockwise.

Joseph I. Pascal.

Dr. Keeler's Heredity Exhibit at the Howe Laboratory

The Heredity Exhibit now set up at the Howe Laboratory depicts the results of a survey of hereditary blindness among pupils of the Perkins Institution, and was prepared through the co-operative efforts of the Perkins Institution with the Howe Laboratory.

The slogan of the exhibit forecasts that "The elimination of hereditary eye defects will be the next great advance in preventive ophthalmology."

A table demonstration classifies, according to the parts affected, 120 hereditary variations of the ocular system, including both harmless and detrimental characters reported in the literature.

A wall chart shows graphically the relative frequencies of occurrence for the 10 most common types of hereditary blindness based principally upon Best's estimates. Other charts show the clinical features of these 10 types of hereditary blindness together with pedigrees depicting their modes of heredity. The pedigrees are chiefly of families represented at Perkins Institution.

Another chart classifies the 1930 enrollment at Perkins Institution into

(1) Causes probably hereditary:	
Optic nerve atrophy	54
Congenital cataract	38
Retinitis pigmentosa	6
Chorioretinitis	5
Infantile glaucoma	3
Progressive myopia	2
Other congenital troubles	88
	196
(2) Causes not hereditary:	
Ophthalmia neonatorum	15
Accidents	12
Phlyctenular keratitis	2
Interstitial keratitis	1
Trachoma	1
Other causes	63
	94

This classification demonstrates the fact that heredity is easily the greatest single cause of blindness at the Perkins Institution. It shows further that cataract and optic nerve atrophy greatly preponderate in the Perkins enrollment just as they do in Best's estimates. Glaucoma, which is commonly developed during adult life, should not be expected among Perkins pupils.

It is evident, then, that war should be declared immediately upon hereditary cataract, glaucoma, and optic nerve atrophy. But what shall be the strategy of the battle?

A chart shows what happened at Perkins Institution when public-minded citizens properly educated to the need, demanded silver nitrate laws. Ophthalmia neonatorum dropped from 18 percent of the enrollment in 1888 to 0.52 percent in 1930. It is then stated that 33 percent of the Perkins enrollment in 1930 suffer from congenital cataract and optic nerve atrophy alone. These two causes, as well as glaucoma, usually behave as Mendelian dominants in heredity. Hence hereditary blindness from its three outstanding causes could be nearly eliminated in a single generation if individuals suffering from them could be restrained from reproduction by an intelligent and active public opinion.

W. Holbrook Lowell,
Boston.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is only mentioned in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy and embryology

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1. GENERAL METHODS OF DIAGNOSIS

Best, F. **On the technique of visual field taking.** Zeit. f. Augenh., 1933, v. 79, Feb., p. 457.

Best does not agree that great refinement of methods of field taking necessarily leads to false results because of the many possible sources of error. Of course the examiner must be familiar with such sources of error. A most important point is constancy of fixation. Best mentions the simplest method to make fixation easier is by projection of the test object. He uses a Bjerrum screen at two meters and does not use objects smaller than three millimeters at two meters, these subtending an angle of five minutes. Whether quantitative perimetry with test objects of various sizes is superior to color perimetry is not clear. In brain lesions he advises both. Although physiologically Ferree and Rand have shown that scotomata are more easily measured with form sense than color sense, it is Wessely's experience that in glaucoma a scotoma is more readily demonstrable with red test objects. It is advisable to determine the blind spot first, done with the projection method even with untrained patients. One should tell the patient from which direction the object is to be expected to appear. For perimetry projection of the test object is not practical and Best recommends the perimeters of Ferree and Rand and of Lauber.

F. Herbert Haessler.

Bömer, M. **On the practical use of Sander's pupilloscope.** Klin. M. f. Augenh., 1933, v. 90, Feb., p. 207.

New tests indicate that this apparatus, although simple and less expensive than Hess's pupilloscope, does not suffice for exact quantitative measurement of the light reaction of the pupil, especially not in the most important cases of incipient pupillary disturbance.

C. Zimmermann.

Feilchenfeld, W. **Diocaine not adapted for tonometry.** Klin. M. f. Augenh., 1933, v. 90, March, p. 393.

Confirming the statement of Ammann (Klin. M. f. Augenh., 1932, v. 89, p. 807), Feilchenfeld observed a severe attack of glaucoma after diocaine. Under eserin and pilocarpin the patient did not regain the previous condition for three months. Beside holocaine, pantocaine and larocaine are serviceable for tonometry, whereas psicaine causes too much burning. C. Zimmermann.

Ferree, C. E., and Rand, G. **A central vision scotometer.** Arch. of Ophth., 1933, v. 9, April, pp. 608-616.

In this communication the authors report a new instrument, simple in construction, for determining the presence of central scotomas, for different degrees of intensity and size as well as for various colors. A greater degree of accuracy is obtained than by other methods. M. H. Post.

Gradle, H. S., and Ackerman, W. **The reaction time of the normal pupil.** Trans. Sect. on Ophth., Amer. Med. Assoc., 1932, 83rd annual session, pp. 342-347. (See Amer. Jour. Ophth., 1933, v. 16, Jan., p. 80.)

Pascal, J. I. **Simultaneous comparison in subjective testing.** Amer. Jour. Ophth., 1933, v. 16, June, p, 509.

Poos, F. **The clinical significance of pharmacodiagnostic reactions on the eye.** Klin. M. f. Augenh., 1933, v. 90, Feb., p. 166.

Pharmacodiagnostic reactions on the eye (ophthalmoreactions in a wider sense), are abnormal reactions to poisons which in their peculiar kind and degree depend on special conditions and processes in the whole organism or parts of it connected with the eye; and hence permit of more or less certain diagnostic conclusions as to pathological processes outside of the eye. Individual and racial differences, hypersensitiveness to medicaments, idiosyncrasies, tuberculin reaction in the eye, ocular reactions in neuroses of the vegetative nervous system and diseases of the endocrines, anisocoria and pharmacological pupillary diagnosis in unilateral morbid processes, and diagnostic adrenalin mydriasis, are discussed in special sections, followed by a chronological bibliography.

C. Zimmermann.

Rowland, W. D. **Visual field studies. IV. Pseudocontractions of upper form field.** Amer. Jour. Ophth., 1933, v. 16, June, p. 496; also Trans. Amer. Acad. Ophth. and Otolaryng., 1932, 37th annual meeting, p. 299.

Vail, H. H. **Vidian neuralgia with special reference to the eye and orbital pain in suppuration of the petrous apex.** Arch. of Otolaryng., 1933, v. 17, Feb., p. 212.

The author explains the ocular and orbital pains typical of petrous suppuration in the early stages, as not due to irritation of the ophthalmic division of the fifth nerve but to irritation of the great superficial petrosal nerve. Anatomical relationships are discussed. The nerve is extradural and there must be dural involvement before the gasserian ganglion and its branches are affected. Three cases of vidian neuralgia are reported to show that irritation of the vidian nerve in the floor of the sphenoid sinus causes ocular and orbital pain. The pains with suppuration of the petrous bone resemble those found in vidian neuralgia.

M. E. Marcove.

Vogt, Alfred, **Contribution to the history of red-free light, with remarks on the alleged claims of priority by Helmbold "on the use of red-free light for examination".** Klin. M. f. Augenh., 1933, v. 90, Feb., p. 225.

In an elucidating survey on the subject Vogt shows that he himself, in 1911, invented the use of red-free light for rendering visible the natural color of the macula, its absence and changes, opacity of the retina, and delineation of its nerve fibers and their alterations.

C. Zimmermann.

2. THERAPEUTICS AND OPERATIONS

Castroviejo, Ramon. **A new knife for ophthalmic surgery.** Amer. Jour. Ophth., 1933, v. 16, April, p. 336.

Davis, F. A. **Tribromethanol (avertin) as an anesthetic in eye surgery.** Wisconsin Med. Jour., 1933, v. 32, Jan., p. 14.

This is practically identical with the paper published in the American Journal of Ophthalmology, 1932, volume 15, page 208.

Flynn, Frank, **Mydriatic synergy. A note on the use of mydriatics by subconjunctival injection.** Brit. Jour. Ophth., 1933, v. 17, May, p. 298.

The author believes that failure of atropin is due to its slow rate of absorption and its low concentration in the aqueous. A synergic mixture of atropin, cocaine, and suprarenin given subconjunctivally has the advantage of entering the aqueous in higher concentration. The atropin paralyzes the sphincter muscle; the suprarenin stimulates the dilator muscle, prevents the solution passing rapidly into the general circulation, and lessens vascular engorgement; and the cocaine aids the suprarenin and renders the mydriasis painless.

The solution, termed "mydricain," contains in each five minim dose atropin sulphate 1/60 gr., cocaine hydrochloride 1/10 gr., and levo-rotatory suprarenin 1/600 gr., together with sodium chloride 1/80 gr. and chorbutol 1/120 gr., in sterile water. The solution is preserved in six minim sterile ampoules. The method is indicated in the initial stages of iritis during the formation of synechiae. It is ineffective in organized synechiae of long standing. The technique of administration consists of first instilling a few drops of four percent cocaine solution, selecting the probably most resistant part of the pupil, for example where there is an obvious synechia, and injecting five minims of the solution subconjunctivally, raising a bleb a few millimeters from the limbus. The rapidity of action is rather startling. Accurate records show that the pupil begins to dilate in from thirty to sixty seconds, reaching its maximum in from three to four minutes. D. F. Harbridge.

Gebb, H. **Pantocain in ophthalmology.** Zeit. f. Augenh., 1933, v. 79, Feb., p. 467.

For one and one-half years the author has used a two percent solution of pantocain for surface anesthesia and a one percent solution with suprarenin for injection. Accommodation and intraocular tension are not affected. One or two instillations do not influence the size of the pupil, but continued use produces some dilation. A two percent pantocain solution is equivalent to a four or five percent solution of cocaine. A one percent solution of pantocain is an adequate substitute for novocain in all ocular surgery.

H. Herbert Haessler.

Haase, G. **Improved glass electrode which can be sterilized for treatment of the conjunctiva, especially in trachoma, with iontophoresis.** Klin. M. f. Augenh., 1933, v. 90, March, p. 391. (Ill.)

The electrode is described and illustrated. C. Zimmermann.

Haldeev, S. I. **On the treatment of ocular diseases without dressings.** Sovietskii Vestnik Opht., 1932, v. 1, no. 12, p. 491.

Haldeev confirms the favorable reports of Russian authors on omission of dressings in postoperative care of eye patients. He performed eighty-one operations, among them thirty-six intraocular, without resorting to eye dressings, and has seen no untoward results. As a rule he applies a palpebral suture for one day, uniting loosely the upper and lower eyelids of the operated eye. M. Beigelman.

Jameson, P. C. **Vascularization of the anterior segment of the eye.** Arch. of Ophth., 1933, v. 9, April, pp. 523-530.

In operating on severe pinguecula, the author's attention was called to the fact that the blood supply arose both from the conjunctival vascular system and deeper ciliary vessels. These last vessels, in fact, form the major part. It was noted further that vessels from this system passed directly under the membrane, that is, the capsule, into the corneal substance. There was little or no anastomosis between the two systems. In a case of vascularization of the cornea a similar blood supply was demonstrable. Curettage and grattage of these deeper vessels were ineffectual. Microscopic examination showed them to have been irritated, but not destroyed. A Graefe cataract knife was therefore used, and seven parallel linear incisions were carried down to the sclera. The vessels bled freely. When the débris was sponged away, the vessels had entirely disappeared, leaving

only the glistening sclera. The author suggests that such a procedure (complete peridectomy) may be of value in connection with the usual glaucoma operations, especially of the congestive type. M. H. Post.

Kochmann, M., and Nabe, J. **Quantitative comparative studies of the action of local anesthetics on the cornea of the rabbit.** Zeit. f. Augenh., 1933, v. 79, Feb., p. 517.

Practically all local anesthetics now in use were studied. The effective concentration of corneal anesthesia, the time of appearance and the duration were measured, and the effect on the pupil and tissues was noted. The lethal dose for guinea pigs was determined and the relation of this quantity to the effective concentration was expressed in a quotient which has practical value in judging a local anesthetic. Pantocain is superior to the others and far better than cocaine. F. Herbert Haessler.

Koman Nayar, K., and Narayanaswamy Nayudu, G. J. **Retrobulbar injections and seventh nerve block.** Brit. Jour. Ophth., 1933, v. 17, April, p. 233.

During the past few years the authors have administered retrobulbar injection and seventh nerve block to over 5,000 patients. At first they followed Wright's method, later they adopted the method of O'Brien as described by Large.

Orbital hemorrhage occurred very rarely and only in the ordinary cataract extraction, not in congested or inflamed eyes. Paralysis of the inferior rectus was encountered frequently, and paralysis of other muscles occasionally. Injections of as much as 4 to 6 c.c. of a two percent solution have been used with no untoward effects.

D. F. Harbridge.

Marquez, M. **Observations on iris operations.** Arch. de Oft. Hisp-Amer., 1933, v. 33, April, p. 204.

The author points out that for optic purposes the sphincter only should be excised, and that the distance of the corneal incision from the center is determined by the amount to be excised. Unless predetermined by the location of the corneal scar, the iridectomy should be nasal. The poorest location is upward. The horizontality or verticality of the corneal incision should be determined by the presence of corneal astigmatism with a view to its neutralization by the postoperative astigmatism. Occasionally the effect of an optic iridectomy is to interfere with binocular single vision. Iridocapsulotomy after extraction with iris prolapse should be done through a horizontal upward incision in order to cut iris meridionally and not, as is recommended, through a vertical lateral incision which cuts across the iris vessels. Iridorexis is preferred through an incision in opaque cornea. For the mechanical purpose of relieving pull on the sphincter by an adherent leucoma, sphincterectomy is done at a point opposite the adhesion. In chronic glaucoma the conjunctival flap is made from the limbus upward (following Dupuy Dutemps), the incision is made with a keratome, and both upper and lower lips are punched out with Lagrange punch forceps. (Illustrated.)

M. Davidson.

Moncreiff, W. F., Coulter, J. S., and Holmquest, H. J. **Experimental studies in diathermy applied to the eye and orbit.** Amer. Jour. Ophth., 1933, v. 16, March, p. 193.

Moreu, A. **Gold salts in ocular therapeutics.** Arch. de Oft. Hisp.-Amer., 1933, v. 33, March, p. 129.

The author reviews the literature and reports sixteen cases treated, including phlyctenular keratoconjunctivitis, rheumatic scleritis, tuberculosis of the lacrimal sac, tuberculous iritis, tuberculous interstitial keratitis, and solitary tubercle of the choroid, and arrives at the following conclusions: Gold salts are as harmless as salvarsan, are always beneficial and often curative in ocular tuberculosis, and are of uncertain efficacy in trachoma. There are cases of intolerance. Dosage is the greatest problem. The effect of subconjunctival injection is rapid and no depo-

sition of gold has been observed. Allocrysin, which can be employed intramuscularly, is the preparation of choice. The indication for gold salts is limited to chronic processes.

M. Davidson.

Neurauter, A. **Ephetonin in ophthalmology.** Klin. M. f. Augenh., 1933, v. 90, April, p. 529.

Ephetonin, synthetic ephedrin, acts on the sympathetic nerve as a sympathicotonic, increases blood pressure, decreases intraocular tension, and is a spastic mydriatic. In from 0.3 to 0.5 percent solutions it produces ischemia of the conjunctiva of longer duration than adrenalin. In three out of five cases of iritis it tore previously unyielding synechiae. Like adrenalin its action on the light iris was much stronger than on the dark-colored. Subconjunctival injections are more powerful and were employed when instillation was not sufficient. C. Zimmermann.

Ruotolo, Giuseppe. **The diffusion of crystalloids in inflamed eyes.** Giornale di Terapia Oftalmologica, 1932, 13th year, nos. 9-10.

The author studied the problem whether crystalloid substances administered intravenously in the course of ocular inflammations tended to undergo elective fixation in the aqueous humor. The substances used were potassium ferrocyanide, urotropin, sodium salicylate, sodium iodide, and copper lactate. The author concludes that the osmosis of these substances into the aqueous humor is not subject to any special electivity. W. H. Crisp.

Tupinamba, Jacques. **Modification of the technique of the conjunctival flap.** Rev. de Ophth. de São Paulo (Brazil), 1933, v. 2, March, p. 174.

After the use of a sliding conjunctival flap in cases of herniation of the iris through the ruptured cornea, the author has seen a good many failures. He criticizes particularly the simple cauterization of herniated iris before making the conjunctival flap, and suggests that failure of the flap to adhere is due to interposition of the burned tissue between the flap and the healthy iris. His modification consists of curetting the cauterized surface of the iris; and after this procedure he has never had to repeat the operation.

W. H. Crisp.

Wolff, J. **Sodium-evipan narcosis in ophthalmology.** Klin. M. f. Augenh., 1933, v. 90, March, p. 385.

Sodium-evipan is a ten percent watery solution of a complicated methyl-barbituric acid combination. From four to six c.c. is injected intravenously in the course of sixty to ninety seconds, producing narcosis for eight to ten minutes. So far 1100 narcoses without accident have been reported.

C. Zimmermann.

3. PHYSIOLOGIC OPTICS, REFRACTION, AND COLOR VISION

Alaerts, L. **The visual field and depth perception.** Arch. d'Opht., 1933, v. 50, April, p. 265.

Stereoscopic vision is rendered possible by the fact that images on the homolateral retinal areas of the two eyes do not occupy corresponding points. Stereoscopic vision is found unimpaired in individuals with a central scotoma but normal peripheral field. On the contrary, those patients possessing only good central vision and a very restricted field have great difficulty in depth perception with a stereoscope. In homonymous hemianopsia, or with test cards using homolateral fields, stereoscopic vision is still possible; but when contralateral fields are tested the image may be fused, but there is no depth perception. The writer approves the definition of Cantonnet and Filliozat for stereoscopic vision, "the cerebral fusion of two images, superimpossible but not identical."

M. F. Weymann.

Bartley, S. H., and Bishop, G. H. **The cortical response to stimulation of the optic nerve in the rabbit.** Amer. Jour. Phys., 1933, v. 103, Jan., p. 159.

The authors describe the cortical response to stimulation of the optic nerve

which can be observed from the cortex. The response consists typically of a series of five major components occurring at intervals of approximately one-fifth of a second. Variations in this series include complexity of components, frequent omission of one or more component, and occasional reversal of the potential sign. M. E. Marcove.

Bartley, S. H., and Bishop, G. H. **Factors determining the form of the electrical response from the optic cortex of the rabbit.** Amer. Jour. of Phys., 1933, v. 103, Jan., p. 173.

This paper presents various lines of evidence as to the character of the electrical responses of the cortex and as to the position and polarity of such elements. The records, however, cannot be interpreted in the same sense as those from isolated nerve and muscle. Results of direct stimulation to an isolated area of the cortex indicate a long refractive period corresponding to the slow rhythm of the spontaneous activity observed. Tying off the blood supply to an area or isolating it by superficial cuts renders it nonresponsive after a few minutes. These procedures demonstrate that cells are involved as well as fibers. Anesthetics depress the activity of the cortex in about parallel degree to depression of reflex function. M. E. Marcove.

Berens, C., Connolly, P. T., and Kern, D. **Certain motor anomalies of eye in relation to prescribing lenses.** Amer. Jour. Ophth., 1933, v. 16, March, p. 199.

Bishop, G. H. **Cyclic changes in excitability of the optic pathway of the rabbit.** Amer. Jour. Phys., 1933, v. 103, Jan., p. 213.

Successive equal stimuli applied to the optic nerve of the rabbit evoke from the contralateral optic cortex responses of variable magnitude, unless the stimuli are very strong or are timed at certain critical intervals. The shortest critical interval of one-third to one-fifth second presumably corresponds to a cyclic spontaneous variation in the accessibility of the optic pathway, of the character of partial refractoriness, but independent of the stimuli applied. Strong stimuli (activating many fibers of the optic nerve) seem to be able to activate such pathways even when partially refractory, as if by summation of the effects of impulses arriving over many optic nerve fibers. This and other similar evidence suggest that the projection pathway from one point of the retina to a corresponding point of the cortex is multiple, and subject to the influence of impulses over other similar pathways. Various experimental and hypothetical considerations are advanced in an attempt to construct a workable scheme that would reconcile these facts with vision.

M. E. Marcove.

Ferree, C. E., and Rand, G. **The eye as a factor in the difference in hue between daylight and twilight.** Amer. Jour. Ophth., 1933, v. 16, June, p. 494.

Granit, Ragnar. **The components of the retinal action potential in mammals and their relation to the discharge in the optic nerve.** Jour. Phys., 1933, v. 77, Feb., p. 207.

Biological analysis of the complex action potential of the retina has been studied by giving the animal ether and by interfering with the blood supply of the retina. The paper is technical in an exaggerated degree and will be incomprehensible to most ophthalmologists.

Green, J., and Beisbarth, C. **Inexpert refraction.** Trans. Sec. on Ophth., Amer. Med. Assoc., 1932, 83rd annual session, pp. 64-79. (See Amer. Jour. Ophth., 1933, v. 16, Jan., p. 74.)

Jackson, Edward. **Aberrations of eyes and lenses.** Trans. Sec. on Ophth., Amer. Med. Assoc., 1932, 83rd annual session, pp. 117-134. (See Amer. Jour. Ophth., 1933, v. 16, Jan., p. 74.)

Kühl, A. **The possibility of determining the refraction subjectively.** Graefe's Arch., 1933, v. 129, p. 455.

In six individuals ranging in age from twenty to sixty-four years, the author tabulated the decimals of vision

by subjective measurement, as modified by lenses varying in strength between +3.00 and —16.00D. With the variables of vision and lens-strength, curves were drawn for comparison. The correction necessary because of diminution of vision for distance resulting here from accommodation is next determined by a formula for lens-strengths between +2.00 and —14.00D. Curves for the six subjects were plotted from the variables of the relative vision and the overcorrection resulting from accommodation. H. D. Lamb.

Lancaster, W. B. **Astigmatic charts versus crossed cylinders: results in one hundred office cases.** Trans. Amer. Acad. Ophth. and Oto-Laryng., 1932, 37th annual meeting, p. 226.

The methods using the astigmatic charts and the crossed cylinders are described in detail. The sensitiveness of both tests depends on the patient's powers of observation. The impression left in the author's mind by the comparison is that both tests are valuable, but that when the best models are used the dials give more prompt, consistent and convincing results in a larger percentage of cases than any other tests for astigmatism. There are a minority of cases, however, where the dials do not yield prompt and consistent results because of irregular astigmatism or poor powers of observation, but where the crossed cylinders are more convincing and consistent. Often in these cases a second trial with the dials after the crossed cylinders have been used will confirm the latter and convince the examiner that he has the correct result. Inasmuch as some patients do better with one method and some with the other, the ophthalmologist should be able to make use of both. (Discussion.)
George H. Stine.

Lemoine and Valois. **Considerations as to etiology and treatment of progressive myopia (ectatic sclerochoroiditis).** Arch. d'Opht., 1933, v. 50, April, p. 246.

The authors believe that malignant or progressive myopia represents a diseased condition of the sclera and choroid. They consider all cases of myopia malignant where the corrected visual acuity is less than two-thirds normal. In the statistics this means that seventeen percent of all myopia is malignant, which approaches the figure of twenty percent in which depigmentation of the choroid was found by Knies. The underlying factors of this ectatic sclerochoroiditis in order of frequency are tuberculosis, syphilis, focal infection in neighboring parts, or disturbance of endocrine glands. The writers recommend careful investigation as to which of these conditions is responsible, and suitable prolonged treatment. Tuberculin or antisyphilitic treatment is given on the slightest evidence of such infection, or even empirically. A table showing initial and final visual acuity of forty-six patients, and duration of treatment, is presented.
M. F. Weymann.

Levinsohn, G. **Experimental proof of the genesis of myopia.** Klin. M. f. Augenh., 1933, v. 90, April, p. 504.

With reference to his experiments for producing myopia in monkeys by the effect of gravity, Levinsohn discusses articles of Essed and Soewarno, Marchesani, and especially the very recent one by Tamura. Tamura's contention that artificially produced myopia in the monkeys was due to increased intraocular pressure is rejected as erroneous.
C. Zimmermann.

Lo Cascio, G. **Refraction with oblique incidence in the eye and the real form of the surface of the crystalline lens.** Ann. di Ottal., 1932, v. 60, Nov., p. 793.

The surface of the cornea is not that of a sphere but is paraboloid. The author's calculations verified by skiascopy lead to the conclusion that the obliquely incidental bundle of rays is slightly astigmatic but rather less so than in a schematic eye of spherical form (Bibliography.) Park Lewis.

Luckiesh, M., and Moss, F. K. **Size of pupil as a possible index of ocular fatigue.** Amer. Jour. Ophth., 1933, v. 16, May, p. 393.

Müller, H. K. **The theory of velonoscopy.** Zeit. f. Augenh., 1933, v. 80, April, p. 113.

The earlier theorists who attempted to explain velonoscopy were in error in that they assumed that the observed object was a point of light or a row of points. The author demonstrates how the velonoscopic shadow arises and why it has the color of the background. The needle transforms the pupil into a screen with double aperture, so that conditions are similar to those in Scheiner's experiment. The cause of the velonoscopic phenomena is the monocular diplopia. It is important to realize that the images are not sharply in focus. The distance between the double images determines the width of the velonoscopic shadow and is dependent on the thickness of the needle, the degree of ametropia, and the size of the image in the focal plane. When a "line" with one wavy edge is used in velonoscopy, the shadow will also be variable in width despite the fact that the needle is straight.

F. Herbert Haessler.

O'Connor, Roderic. **Corneal cautery for high myopic astigmatism.** Amer. Jour. Ophth., 1933, v. 16, April, p. 337.

O'Connor, Roderic. **Lens removal for high myopia.** Amer. Jour. Ophth., 1933, v. 16, June, p. 516.

Richmond, P., and Ebert, E. C. **Speed of visual perception.** United States Naval Med. Bulletin, 1933, v. 31, April, p. 150.

By placing before both eyes a camera shutter for measuring speed, the authors determined the length of time necessary to recognize letters or colors. Three hundred men were examined, all with vision of at least 20/20. The authors conclude that a time factor should be incorporated in visual acuity records, as some patients are able to improve vision by lid squeezing if given sufficient time. The visual speed ranged from 0.5 to 0.01 second per letter, the former being a good standard unit for each eye. The speed of perception for both eyes was usually twice that of monocular vision. Color perception was not diminished so rapidly as form perception on shortening the period of observation.

M. E. Marcove.

Rossi, G. **Concerning the duration of the act of accommodation in the pregnant.** Arch. di Ottal., 1933, v. 40, Jan., pp. 17-31.

The author used pregnant women with normal vision, having emmetropia and normal amplitude of accommodation. His experiments did not demonstrate that the state of pregnancy exercised any influence on the time of accommodation. Herman D. Scarney.

Rutherford, C. W. **Asthenopia, a psychoneurosis and a medical responsibility.** Trans. Sec. Ophth., Amer. Med. Assoc., 1932, 83rd annual session, pp. 33-46. (See Amer. Jour. Ophth., 1933, v. 16, Jan., p. 74.)

Smith, Dorland. **Practical cyclodamia.** Amer. Jour. Ophth. 1933, v. 16, April, p. 319.

Tocantins, L. M., and Jones, H. W. **Defective color vision and its handicap in medicine.** Amer. Jour. Med. Sciences, 1933, v. 185, Feb., p. 243.

The authors report that nine out of seventy junior medical students showed defective color vision. Difficulties encountered in training for practice are mentioned. It is suggested that dermatology is a poor specialty for color defective physicians, while in roentgenology this abnormality might even be an advantage because of the tendency to good discrimination of brightness values. (Four references.)

Ralph W. Danielson.

Zanettin, G. **The chromatic sense in indirect vision.** Ann. di Ottal., 1932, v. 60, Dec., p. 873.

The author, who studied indirect radiation with modern and scientifically constructed instruments, finds that chromatic sensibility decreases rapidly from center to periphery but more for

red and green than for blue. To four degrees in the perimacular region the sensibility for red decreases less rapidly than for blue or green, and reduction is less rapid in the nasal segment than in the temporal, the superior, and the inferior in the order named. It also varies between one subject and another. (Bibliography) Park Lewis.

4. OCULAR MOVEMENTS

Duane, Alexander. **Binocular movements.** Arch. of Ophth., 1933, v. 9, April, pp. 579-607.

This paper is a further abstract from the author's uncompleted book on muscles of the eye. As with previous chapters, it does not lend itself to abstraction. The subject is treated first from the standpoint of the general character and purpose of such movements, secondly, the conjugate or parallel movements, and thirdly the vergences. M. H. Post.

Hosford, G. N., and Hicks, A. M. **Two improved instruments for use in the O'Connor cinch shortening operation for heterotropia and heterophoria.** Amer. Jour. Ophth., 1933, v. 16, June, p. 528.

Houser, K. M. **Infection of the sphenoid sinus as a possible cause of abducens paralysis.** Pennsylvania Med. Jour. 1933, v. 36, March, p. 411.

The author studied a series of nineteen heads to determine anatomically the relationship between the sphenoid sinus and the abducens nerve. He found that there was only an interval of 2 mm. between these two structures. The sphenoid may account for some abducens paralyses otherwise unexplained. M. E. Marcove.

Luhr, A. L., and Eckel, J. L. **Fixation and voluntary nystagmus.** Arch. of Ophth., 1933, v. 9, April, pp. 625-634.

The authors report two cases, one of fixation and one of voluntary nystagmus. Both fall under the ocular category. In the first case nystagmus was induced by fixing steadily on any point in the visual field. Rapid oscillation was followed by sharp convergence, pain, and closure of the lids, with immediate return to normal. In the second case voluntary nystagmus was produced by concentrating on a desire to make the eyes dance. Thirty seconds is about the time limit for this type. M. H. Post.

Marlow, F. W. **Recent observations on the prolonged occlusion test.** Amer. Jour. Ophth., 1933, v. 16, June, p. 519.

O'Connor, Roderic. **Superior rectus shortening to correct certain horizontal deviations.** Amer. Jour. Ophth., 1933, v. 16, May, p. 432.

Ohm, J. **The opticokinetic reaction in congenital nystagmus.** Graefe's Arch., 1933, v. 129, p. 526.

Congenital nystagmus was generally associated with a disturbance of opticokinetic nystagmus. This was slight in part of the cases and consisted of a diminution of its frequency and amplitude, sometimes to the point of more or less complete suspension which the author designated as opticokinetic rigidity of rotation. Another frequent disturbance was so-called inversion or reversal of direction of motion. In many cases the opticokinetic reflex was at times regular and at other times acted abnormally. There existed no relation between the organic condition of the eye and the variety of disturbance of the opticokinetic reflex. H. D. Lamb.

Peter, L. C. **The treatment of nonparalytic squint.** Amer. Jour. Ophth., 1933, v. 16, June, p. 481; also Trans. Ophth. Soc. United Kingdom, 1932, v. 52, p. 325.

Poe, D. L. **Ophthalmoplegia totalis.** Amer. Jour. Ophth., 1933, v. 16, June, p. 512.

Van der Hoeve, J. **Bowman lecture of 1932. Ocular movements.** Trans. Ophth. Soc. United Kingdom, 1932, v. 52, pp. 1-31.

The author writes of the complicated nature of ocular movements; he shows

that there is no one single center of rotation of the eye and that for every departure from the primary position the center of rotation changes; and that lenses such as punctal, ground on the principle that such a center is present, are sometimes found less satisfactory than biconvex and biconcave.

Thomas D. Allen.

5. CONJUNCTIVA

Alvaro, M. E. **Ophthalmia neonatorum in the Santa Luzia Dispensary (São Paulo).** Rev. de Ophth. de São Paulo (Brazil), 1933, v. 2, March, p. 185.

The author especially recommends injection of sterile milk in doses of 1 to 2 c.c., repeated every day or every second day according to the individual reaction. He does not feel that the substitutes for milk can compete with it as to efficiency. W. H. Crisp.

Barrie, T. S. **A rare disease of the conjunctiva with spontaneous cure.** Glasgow Med. Jour., 1933, v. 1, Feb., p. 33.

A man of twenty-one years came because of defective vision. There was a slight degree of enophthalmos of the left eye with increased action of the left occipitofrontalis muscle. Upon everting the upper lid of this eye a well defined reddish layer slipped forward as far as the cornea. A similar appearance was noted in the lower fornix. Biopsy and microscopic examination showed the tissue to be altered conjunctiva. The squamous epithelium was thickened and showed many goblet cells, and there were many lymph nodes separated by strands of connective tissue beneath the epithelium. Eight months later the conjunctiva was entirely normal. The patient was a brush fiber mixer by occupation, and the condition was probably due to a fiber or bristle which had been in the fornix for a long time.

M. E. Marcove.

Cordero, C. **The behavior of hemoisoagglutins in trachoma.** Ann. di Ottal., 1932, v. 60, Oct., p. 748.

As determined by their agglutination reactions there are four blood groups. The mass of Europeans are found in groups one and two. The author studied the behavior of blood groups in 210 cases of trachoma, and found a biochemic index of 3.5, differing but little from the normal (3.6). This difference is doubtless due to the larger number of cases found in group two. In the complicated forms such as pannus and xerosis the biochemic index descends to 2.83, chiefly by augmentation of group 4, rarely found in occidentals.

Park Lewis.

Cuénod and Nataf, R. **Concerning the contagiousness of trachoma; three new cases of direct contagion.** Arch. d'Opht., 1933, v. 50, April, p. 261.

Three physician confrères known to the writers had received definite trachomatous infection by transmission of trachomatous material from patients' eyes into their own at operation. In one case the infection was bilateral, in the other two it remained unilateral, (see editorial, American Journal of Ophthalmology, 1933, volume 16, page 545.) M. F. Weymann.

Derkač, V. **Experiences with intravenous injections of solutions of ammonium-copper sulphate in trachoma.** Klin. M. f. Augenh., 1933, v. 90, April, p. 532.

Derkač reports his experiences with three hundred injections. They showed a rapid symptomatic effect in relieving photophobia, lacrimation, and blepharospasm, but had no specific influence on trachoma. C. Zimmermann.

El-Bakly, M. A. **Trachoma—some clinical observations.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 77-91.

Clinical observations made during three years of routine examination and treatment of trachoma in the primary schools of Egypt are given by the author. The MacCallan classification of trachoma is used in the presentation of his findings. The article begins with a description of the normal conjunctiva and attention is called to the fact that in many young children small lymph

follicles are normally seen in the upper and lower fornices or at the angles. A differential diagnosis of normal follicles, follicular conjunctivitis and trachoma is given in tabular form. T. 1 is considered as a fairly late rather than an early stage of trachoma. The disease begins in the lower fornix and later appears in the upper tarsus. In many cases the lower lid shows the typical appearance of T. 2a, before any follicles of T. 1 are seen on the upper lid. Nearly 5/6 of early trachoma cases remain dormant as T. 1 for years. The difficulty in diagnosing trachoma is experienced only in its early stage before the follicles appear on the upper tarsus. Tricoire's reaction and vital staining with methylene blue have proved helpful in the diagnosis of early cases. Type T. 2b (papillary hypertrophy coexisting with follicles) is considered a complication of trachoma and not an original type. The article is concluded by an enumeration of the various types of change which the cornea may show in trachoma. Edna M. Reynolds.

Grasz, E. **On trachoma.** Zeit. f. Augenh., 1933, v. 79, Feb., p. 470.

This essay is an evaluation of the work of others and a rather general expression of opinion of the author. Silver nitrate and copper sulphate are the most powerful agents. Expression of follicles and massage of the hypertrophied conjunctiva hasten healing. The author is gratified that excision of the retrotarsal fold is being abandoned. Extirpation of the tarsus is done rarely now, probably because neglected trachoma is more rarely seen.

F. Herbert Haessler.

Howard, H. J. **Results of recent investigations in the etiology of trachoma.** Amer. Jour. Ophth., 1933, v. 16, March, p. 218; and Trans. Amer. Acad. Ophth. and Otolaryng., 1932, 37th annual meeting, p. 81.

Krause, A. C., and Chan, E. **The chemical constitution of the conjunctiva, choroid, and iris.** Amer. Jour. Phys., 1933, v. 103, Feb., p. 270.

This is a detailed chemical study of the conjunctiva, choroid, and iris. Tables show the chemical analysis with special emphasis on insoluble proteins.

M. E. Marcove.

Maitra, J. N. and Sanyal, S. **Epidemic unilateral conjunctivitis with corneal infiltration and adenitis.** Jour. of the Indian Med. Assoc., 1932, v. 2, Nov., p. 101.

In this epidemic there was a velvety appearance of the palpebral conjunctiva with many opaque granulations irregular in size and distribution. The cornea showed innumerable pin-point infiltrations. Adenitis occurred without suppuration. The condition occurs in the tropics, is more prevalent in the autumn and rainy season, and lasts one to four weeks. In some cases a gram-negative bacillus growing on blood agar only was found. No bilateral cases were seen, there was no rise of temperature, and eosinophilia was present. (Two figures in color, several tables, thirteen references.)

Ralph W. Danielson.

Motegi, A. **Contribution to a kind of microorganism in trachomatous tissue.** Klin. M. f. Augenh., 1933, v. 90, March, p. 378. (Ill.)

Ochi and Nogami previously reported (Klin. M. f. Augenh., v. 86, p. 309) on a kind of microorganism in trachomatous tissue. Motegi's assistant, Fujimura, investigated pure cultures after Ochi's method and confirmed his observations. They are roundish, of a diameter of from 0.8 to 6 micra. Neither pure cultures nor recent trachomatous tissue introduced on the human conjunctiva gave positive results.

C. Zimmermann.

Rumiantzev, A. V., and Levkoieva, E. F. **Observations on the growth of trachoma tissue in vitro.** Sovietskii Vestnik Opht., 1932, v. 1, no. 12, p. 527.

In twenty-two cases of trachoma attempts were made to study tissue cultures prepared according to Maximov's and Carrel's techniques. In the author's experience trachoma tissue always produced an extreme and characteristic

liquefaction of the plasma used for cultures. The trachoma follicle was found to consist in vitro of (1) small and large lymphocytes, (2) normal and hypertrophied monocytes, and (3) normal macrophages. These cells in time degenerated without tending to become transformed into fibrocytes or macrophages. This, in the author's opinion, proves that the follicles are a specifically altered tissue and not a part of a proliferative granulomatous infiltration. The conjunctival epithelium displays in vitro considerable motility and capacity for intense growth, maintaining all its cytotypical characteristics.

M. Beigelman.

Sobhy Bey, M. **The true nature of post-trachomatous degenerations.** Bull. Ophth. Soc. Egypt., 1932, v. 25, pp. 56-59.

The author gives a histopathological description of the so-called degenerated trachoma follicles which appear in the later stages of the disease. These are in reality epithelial downgrowths resembling glands which are transformed into cysts filled with inspissated material that may undergo calcareous degeneration. The author suggests the term "post-trachomatous concretions" to replace the incorrect term "post-trachomatous degenerations". (Discussion by R. P. Wilson with microphotographs.) Edna M. Reynolds.

Soleiman Bey, Sayed A. H., and Attiah, M. A. H. **A comment on the treatment of trachoma by ether-benzyl-cinnamic.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 180-190.

Clinical notes and comments on nine cases of trachoma treated by intramuscular injections of ether-benzyl-cinnamic are given. This method of treatment was introduced by Jacobson of Paris who reported cures of many pathological conditions in which the dominant lesion is lymphoid proliferation. During the administration of "Jacobson's solution" no local treatment was given. The injections were well tolerated, giving neither local nor general reaction, but the results of the treatment were disappointing. During the treatment and afterward there was no diminution in size of the follicles and no regression of the vessels or of the infiltrations in the cornea. This method cannot in any way replace the usual local treatment. Edna M. Reynolds.

Sorour, M. F. **The symptomatic treatment of spring catarrh with injections of splenic extract.** Bull. Ophth. Soc. Egypt, 1932, v. 25. pp. 60-62.

In Egypt there is an endemic form of splenomegaly associated with bilharziosis. Active bilharziosis is ordinarily accompanied by an eosinophilia but cases of active bilharziosis complicated by splenomegaly frequently show leucopenia and very low eosinophilia or none. If splenectomy is done, the eosinophiles begin to appear in the blood stream. From observation of these phenomena, the author concluded that the hyperactive spleen has an inhibiting effect on the production of eosinophiles, and the idea occurred to him to use a splenic extract in the treatment of spring catarrh. Results obtained with twenty cases have been such that after two to eight subcutaneous injections the symptoms have entirely disappeared.

Edna M. Reynolds.

Souter, W. C. **Papillomatosis conjunctivae: spontaneous cure of a case.** Trans. Ophth. Soc. United Kingdom, 1932, v. 52, pp. 32-46.

Two colored illustrations showing the gross and the microscopical appearance accompany this paper. In the discussion primary obstruction of the lymphatics was mentioned as a probable cause. Thomas D. Allen.

Spillmann, J., and Rousseau, F. **Ulcero-vegetating conjunctivitis with voluminous adenopathy cured by methylic antigen.** Ann. d'Ocul., 1933, v. 170, April, pp. 333-336.

A twenty-two-year-old woman presented a large ulcerating mass in the right upper lid and enlargement of the preauricular gland. The Wassermann was negative. The mass was not proved to be tuberculous, but this diagnosis being most likely in view of the general findings the use of methylic antigen of

Nègre and Boquet was started. An intense general reaction occurred, with fever for about three weeks, at the end of which time there was great improvement. With more cautious use of the antigen the condition was cured.

H. Rommel Hildreth.

Thygeson, Phillips. **Inoculation of the human conjunctiva with trachomatous materials.** Amer. Jour. Ophth., 1933, v. 16, May, p. 409.

Tobgy, A. F. **Intravenous injections of copper salts in the treatment of trachoma.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 191-194.

A short summary of the various kinds of copper salts which have been used intravenously is given and twenty cases treated with an organic combination of copper and calcium are reported. A course of twelve injections was given intravenously at three-day intervals. In most cases a definite improvement in the subjective symptoms was obtained but this was only temporary. There was no sign of regression of the follicles and the conjunctiva remained just as congested as ever. Cases with pannus failed to show improvement and some relapses occurred during treatment. Four-fifths of the cases showed definite reduction in the number of red blood cells, in some cases as high as seventeen to twenty percent. In general the results were not so effective as from simple local application of copper.

Edna M. Reynolds.

Tobgy, A. F. **Spring catarrh in Egypt.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 63-76.

A brief review of the various theories regarding the etiology of spring catarrh is given, followed by an analysis of 133 cases treated at the Giza Memorial Ophthalmic Laboratory. Contrary to the general opinion that spring catarrh is a benign disease in attacking the cornea, the author quotes cases of kerato-ectasia and opacity of the cornea resulting from the malignant or hyperplastic type of the disease. In many of these cases it is difficult to differentiate spring catarrh from trachoma, and the slitlamp is valuable in making this differential diagnosis. Discouraging results are reported with surgery, autohemotherapy, splenic extract, arsenicals, calcium, ultraviolet light, and lactic acid. Some satisfactory results followed the use of chaulmoogra oil and 14 out of 25 cases showed definite improvement after the use of radium. (Extensive bibliography.)

Edna M. Reynolds.

Vele, M. **The nuclear inversion of Velez in trachoma.** Ann. di Ottal., 1932, v. 60, Oct., p. 713.

According to Arneth an examination of the blood of the tuberculous will show inversion of the binucleated and trinucleated cell; the trinuclear neutriphiles being in the majority in the normal blood, while in the tuberculous the binuclear are greater in number. Velez found this inversion only in atypical incipient tuberculosis. The author examined the blood of 125 trachomatous cases with a view to determining whether this inversion held good in trachoma, thinking thereby to establish a relationship between trachoma and tuberculosis. The examinations showed no such inversion to exist, the proportion of neutrophiles being much the same as in normal conditions. (Bibliography.)

Park Lewis.

6. CORNEA AND SCLERA

Abou-El-Fadl, M. **A case of the mild type of dystrophia epithelialis corneae.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 109-114.

A case of epithelial dystrophy of the cornea in a young man aged twenty-six years is reported. The slit-lamp findings are given in detail. The patient's family history was negative and no cause either local or general could be found to explain the dystrophy. The condition improved under treatment with 2.5 percent scarlet red ointment. A brief comment regarding theories of causation, differential diagnosis, and various types of treatment of epithelial dystrophy is appended.

Edna M. Reynolds.

Attiah, M. A. H., and Sobhy Bey, M. **Spring catarrh of cornea.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 103-108.

A case of spring catarrh in which the upper half of both corneae was involved is presented. The patient, a female aged nine years, had been under observation and treatment for four years. The treatment here reported extended over two months. The left eye was operated by shaving the granulations off the surface of the cornea and covering the wound with conjunctival flaps. No operative procedure was undertaken on the right eye but the patient was given a course of splenic extract injections. Both corneae showed even, smooth opacities at the completion of treatment. A description of the microscopic appearance of the tissue removed from the left eye is given as well as a discussion of the histopathology of spring catarrh. Edna M. Reynolds.

Aust, O. **Nummular keratitis of Dimmer.** Graefe's Arch., 1933, v. 129, p. 576.

Between December, 1924, and the end of 1931, twenty-seven cases of this condition were observed in the Second Eye Clinic of Vienna. As had been mentioned by Salzmann, the condition was much more prevalent among those in agricultural pursuits. It was more prevalent in individuals between fifteen and thirty years of age but was observed in patients of all ages between ten and fifty-five years. Patients with this condition applied most frequently for relief between June and October. It was nearly always unilateral. The affected eyeball was usually free of injection but the cornea presented, more frequently at its middle, small superficial foci, on an average ten in number. These foci occurred in three stages of development: (1) a densely gray, disc-shaped, circular opacity 0.5 mm. in diameter, over which the surface was elevated and much stippled; (2) a circular disc 1 by 1.5 mm. in diameter with frequently a densely white central opacity and a gray opaque border, while the disc itself might present all shades of color from a delicate yellowish-gray through gray to bluish-gray, over which the surface was flat and stippled; (3) the facet, characterized by many round flat depressions of the surface which, at first a delicate gray, gradually changed to a bluish tone. Examination with the slitlamp proved that the opacity of a single focus was confined to the anterior third of the cornea, and only the central portion of the opacity might extend to the middle layers of the cornea. A period of two to four months was generally necessary for development of the foci through their three stages. The more common history was that of a mild injury or minute foreign body, then photophobia and tearing had begun, four to six weeks before consulting the clinic, and recently a disturbance of vision had been noticed. In the majority of cases vision was reduced to 6/18, rarely to counting fingers at a few meters. Repeated attempts to find an organism were unsuccessful, and etiology could only be conjectured.

H. D. Lamb.

Bedell, A. J. **Mooren's ulcer.** Amer. Jour. Ophth., 1933, v. 16, April, p. 289; also Trans. Amer. Ophth. Soc., 1932, v. 30, p. 165.

Clapp, C. A. **Additional experiments verifying the presence of treponema pallidum in the cornea in experimental interstitial keratitis.** Amer. Jour. Ophth., 1933, v. 16, May, p. 397; also Trans. Amer. Ophth. Soc., 1932, v. 30, p. 212.

Custodis, Ernst. **Blue sclera and scleral staphyloma.** Graefe's Arch., 1933, v. 129, p. 482.

The parents of the child stated that at five years of age they had observed for the first time in the sclera of the right eye a black spot the size of the head of a large pin. Neither injury nor inflammation had occurred in the eye. When first examined by the author at the age of ten years, there was moderate blueness of both scleras. In the right eye, above and nasally about 2 mm. from the corneal margin, there was a prominent dark colored swelling of the sclera. Under local anesthesia,

cauterization of the staphylomatous formation was done before transplanting a piece of fascia lata and fixing it with two sutures upon the sclera under the conjunctiva. The result was very satisfactory. No other member of the family showed blue scleras.

H. D. Lamb.

Davis, W. T. **Superficial parenchymatous punctate keratitis.** Trans. Amer. Acad. Ophth. and Oto-Laryng., 1932, 37th annual meeting, p. 115.

This form of keratitis is usually of toxic origin and is characterized by periodicity, symptoms of ocular irritation, and conjunctival congestion. It involves the parenchyma but not the epithelium. The characteristic lesions consisting of fine white angular points in the corneal substance, immediately beneath, but not involving the epithelium, can be seen only with the biomicroscope. They stain brilliantly with fluorescein. The left eye was involved in nine out of eleven cases, and in the remaining two both eyes were involved. The local treatment consisted of holocain, mydriatics, rest, avoidance of light, and the use of the thermophore at a temperature of 110° F. for sixty seconds. In eight cases the condition was apparently due to infection in the tonsils and teeth or both. Of the remaining three, one was due to gastrointestinal infection, one to possible tuberculosis, and the third disappeared before a study could be made. Removal of the foci of infection proved efficacious in nearly all the cases. (Two figures and discussion.)

George H. Stine.

Derkač, V. **Treatment of spring catarrh.** Klin. M. f. Augenh., 1933, v. 90, Feb., p. 231.

A farmer aged seventeen years had trachoma, iritis (from hereditary lues?), and spring cattarrh, with pale avascular gelatinous nodules at the corneal limbus of each eye. Although the blood tests for syphilis were negative, inunctions with mercurial ointment cured the iritis and completely removed all changes produced by the spring catarrh. Whether this may be considered as a heredoluetic symptom must be confirmed by further observations.

C. Zimmermann.

Fuchs, Adalbert. **Concerning unusual ulcers of the cornea and their treatment.** Brit. Jour. Ophth., 1933, v. 17, April, p. 194.

The author reports some rather unusual types of ulcer, partly on account of the symptoms, and partly for the success of the therapy. Keratomycosis fascicularis the author has observed only once, and reports are very infrequent in the literature. In the case reported the ulcer had a fine granular mass in the center, and microscopic examination showed mycotic threads. Marasmic ulcer occurred in a patient of seventy-three years. The eye had been operated upon for cataract twenty years earlier. In two cases of rodent ulcer the outstanding feature as to therapy was repeated paracentesis.

The author discusses quite fully three cases of dendritic keratitis. If the usual procedures are not helpful he cauterizes the ulcer with a copper stick, carefully removing the degenerated epithelial layer. Because of the dense scar resulting, this treatment is carefully restricted in its application, but if the ulcer is peripheral it may be used more liberally.

Herpes corneae is rare in children. The last case of two such which the author has observed had a typical dendritic ulcer and a herpetic ulcer in the nasal upper quadrant. It was treated with Bucky's rays to no advantage but responded immediately to the use of copper cauterization. Four illustrations, four photomicrographs.

D. F. Harbridge.

Fuchs, A. **Hydropic swelling of the corneal epithelium.** Klin. M. f. Augenh., 1933, v. 90, March, p. 300. (See Section 7, Uveal tract, sympathetic disease, and aqueous humor.)

Hofe, K. vom. **Does treatment of congenital lues prevent parenchymatous keratitis? Parenchymatous keratitis after trauma.** Klin. M. f. Augenh., 1933, v. 90, April, p. 492.

Four cases are reported in which specific treatment did not prevent parenchymatous keratitis after many years. In the fourth case a short relapse of parenchymatous keratitis occurred after injury of the cornea by a foreign body. Thus the antiluetic treatment eleven years previously had no prophylactic effect. C. Zimmermann.

Judd, J. H. **Nodular degeneration of the cornea.** Amer. Jour. Ophth., 1933, v. 16, April, p. 310.

Krause, A. C. **The chemistry of the sclera.** Amer. Jour. Ophth., 1933, v. 16, March, p. 214.

Kredbova, Pavla. **Metastatic gonorrheal keratitis.** Oft. Sbornik, 1932, v. 7, pp. 238-241.

January 15, 1932, one month after a urethral gonorrheal infection, a nineteen year old male developed bilateral purulent conjunctivitis with negative smears and cultures. Later, superficial punctate central keratitis developed, acutely exacerbated by an injection of gono-vaccine. Subsequent injections of the vaccine were followed by speedy improvement. (Bibliography.)

G. D. Theobald.

Kreiker, A. **Complete covering of the cornea by conjunctiva in rodent ulcer.** Klin. M. f. Augenh., 1933, v. 90, March, p. 373. (Ill.)

Three cases of rodent ulcer were cured by covering the whole cornea with a conjunctival flap shifted from the upper portion and sewed below the lower limbus. After three or four months the flap at the center of the cornea was lifted in a fold and incised with scissors. By the radial tension a round hole of 2 mm. was formed in which the clear cornea, covered with epithelium, and the pupil and iris were visible. C. Zimmermann.

Lane, L. A. **Radium in lesions of the cornea.** Arch. Physical Therapy, X-ray, Radium, etc., 1933, v. 14, Feb., p. 99.

Among the benign lesions responding to radium are ectasias, fistulae, vascular keratitis, vesicular keratitis, keratitis rosaceae, tuberculosis involving the cornea, posterior keratitis, and ulcerative keratitis in its various forms with or without hypopyon. One of the greatest fields of usefulness is the thinning of leucomas and opacities of the cornea, and the earlier radium is used the better the result. In malignant lesions radium often offers better results than surgery alone, there being less loss of tissue. Frequent biomicroscopy is necessary to obtain the best results. (Discussion.) M. E. Marcove.

Mayou, M. S. **Congenital absence of the cornea in a foal.** Trans. Ophth. Soc. United Kingdom. 1932, v. 52, pp. 57-63.

Both corneas were absent. The foal was from famous racehorse parents, and no previous deformities in the family were known. The eyes were round and white, the sclera replacing the cornea. Where the limbus should be, pigmentation developed and gradually advanced over what should have been the cornea. The animal was killed at six weeks. Microscopically the surface epithelium was normal. Bowman's membrane was partly present in one eye. The substantia propria was reduced in thickness and was vascular. No Descemet's membrane was found. Some acinous glands were found in the anterior chamber, attached to the cornea and to a pseudociliary body. No lens was present. There was a congenital detachment of the retina, but no defect of the ocular cleft. The author thinks extrusion of the lens occurred in very early fetal life through perforation of the cornea. Thomas D. Allen.

Reid, A. C. **A series of one hundred cases of hypopyon ulcer, with special regard to a method of treatment.** Brit. Jour. Ophth., 1933, v. 17, March, p. 162.

In this contribution Reid makes certain comparisons with a series of one hundred cases which he had previously reported. In the previous report he had argued for early section of the cornea, but he has since abandoned early section, and now uses a Wessely cautery with certain improvements allowing of application at an exact temperature at the point (between 70° and 90° C.). The

author advises 70° to 85° as the optimum. A few seconds suffices for the point to be passed over the surface, exploring all edges.

Each of the last series when first seen had a hypopyon visible to the naked eye. The two series are compared. D. F. Harbridge.

Riegel, E. **Familial degeneration of the cornea.** Klin. M. f. Augenh., 1933, v. 90, April, p. 498.

Five cases of degeneration of the cornea of unknown etiology are described in members of the same family, observed for three generations. Two cases were seen in earliest youth, suggesting the assumption that familial degeneration of the cornea may be congenital. C. Zimmermann.

Ros, A. **Parenchymatous keratitis.** Arch. de Oft. Hisp.-Amer., 1933, v. 33, March, p. 163.

Ros finds that parenchymatous keratitis, with rare exceptions, is luetic, and that antiluetic treatment is always indicated, combined with local application of steam, atropin and dionin. He rejects subconjunctival injections, methylene blue, and yellow oxide of mercury. M. Davidson.

Sabata, J. **Parenchymatous keratitis following injury.** Oft. Sbornik, 1932, v. 7, pp. 107-108.

The left eye of a man aged forty-four years was injured during an explosion of steam. Two months later he developed in this eye a parenchymatous keratitis, which was regarded as an anaphylactic reaction secondary to lues acquired two years previously. After a course of antiluetic treatment, the eye regained normal vision.

G. D. Theobald.

Salvati, G. **Treatment of staphyloma of the cornea by galvanocautery.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 46-50.

In certain cases of corneal staphyloma, where patients had refused enucleation or amputation of the anterior segment, the author has secured a decrease in the ocular tension and a reduction of the staphyloma by using the Paquelin cautery to destroy the ciliary processes in one or more regions, depending upon the size of the staphyloma. A brief summary of five cases is given. Edna M. Reynolds.

Sanyal, Saridindu. **Epidemic superficial keratitis.** Amer. Jour. Ophth., 1933, v. 16, May, p. 390.

Thomas, J. W. T. **Transplantation of scleral tissue on to rabbit's cornea.** Trans. Ophth. Soc. United Kingdom, 1932, v. 52, pp. 64-73.

Five trephined transplantations were made. All grew, some without vascularization. Several became somewhat transparent. Thomas D. Allen.

Tobgy, A. F. **Some practical reflections on tattooing of the cornea with platinum chloride.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 42-45.

Suggestions regarding technique are given, supplementing an article on the same subject published by the author in volume 24 of this publication in 1931. Preoperative study with the slitlamp is recommended to determine the thickness of the cornea, the presence of vessels, and so on, as well as to determine the width of the nebulous zone surrounding the leucoma. The author has found that this nebulous zone of the cornea must be tattooed as well as the leucoma. Otherwise, a whitish ring surrounds the jet-black tattooed area. Procedure is outlined for tattooing atheromatous and facetted scars in which an even coloration was not obtained at the first operation. Platinum chloride has been found superior to gold chloride for tattooing because it is much less irritating to the cornea and gives a jet-black color which has remained permanent in cases under observation for two years.

Edna M. Reynolds.

Viallefont, H. **The blue-sclera syndrome.** Ann. d'Ocul., 1933, v. 170, April, pp. 289-328.

This review is divided into three parts: (1) A review of the literature.

(2) A description of the clinical aspects of the disease, in which "brittle bones" is so prominent a feature. (3) The basis of the disease, which is definitely hereditary and involves calcium metabolism. There are findings which make treatment directed toward the parathyroids rational. (Extensive bibliography.) H. Rommel Hildreth.

Vorobiev, I. F. **"Chauffage" in the treatment of corneal ulcers.** Sovietskii Vestnik Opht., 1932, v. 1, no. 12, p. 535.

"Chauffage" is a method of thermotherapy proposed by Weekers (Archives d'Ophtalmologie, 1919). A platinum cautery point at white heat is held for one minute at a distance of 2 mm. from the corneal surface and this is repeated three times at short intervals, during which time the cornea is kept moist. Simple corneal ulcerations, including the diplobacillary ones of serpiginous character, respond well to this method of treatment. In severe pneumococcus ulcers and in those of mixed origin the actual cautery must be substituted. M. Beigelman.

Weeks, J. E. **Treatment of corneal infections.** Amer. Jour. Ophth., 1933, v. 16, April, p. 293; also Trans. Pacific Coast Oto-Ophth. Soc., 1932, 20th annual meeting, p. 65.

Wilson, R. P. **A short slitlamp study on the corneal vessels in Egyptian trachoma with a discussion of their diagnostic value in doubtful cases.** Bull. Ophth. Soc. Egypt, 1932, v. 25, pp. 92-102. (See Amer. Jour. Ophth., 1933, v. 16, Jan., p. 80.)

7. UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Baer, Ilse. **Autohemic injections into the anterior chamber in iris tuberculosis and the possibility of controlling the results by animal experiments.** Klin. M. f. Augenh., 1933, v. 90, April, p. 485.

Tubercle bacilli introduced into the anterior chamber of rabbits were in no way influenced in their virulence and the course of the disease was not arrested. It was known from the start that this primary very virulent infection of the animal eye was entirely different from the secondary relatively benign tuberculosis of man, so that the prospect of a therapeutic effect from the injections was very small. The favorable results observed at Schieck's clinic proved that for any kind of chronic tuberculosis of the iris in man autohemic injections are the method of choice. (See also Amer. Jour. Ophth., 1933, v. 16, April, p. 350.) C. Zimmermann.

Bedell, A. J. **Significance of choroidal hemorrhage.** Trans. Sec. on Ophth., Amer. Med. Assoc., 1932, 83rd annual session, pp. 147-170. (See Amer. Jour. Ophth., 1932, v. 15, Dec., p. 1198.)

Benedict, W. L., and Goeckerman, W. H. **Foreign proteins and gold in treatment of uveitis.** Trans. Sec. on Ophth., Amer. Med. Assoc., 1932, 83rd annual session, pp. 237-245. (See Amer. Jour. Ophth., 1932, v. 15, Dec., p. 1198.)

NEWS ITEMS

News items in this issue were received from Drs. L. Waller Deichler, Philadelphia; Walter L. Hogan, Hartford, Conn.; J. W. Jervey, Jr., Greenville, S.C.; and W. Holbrook Lowell, Boston.

News items should reach **Dr. Melville Black, 424 Metropolitan Building, Denver,** by the twelfth of the month.

Deaths

Dr. John Howard Harter, Seattle, aged forty-five years, died, May 13th.

Dr. Henry Dickson Bruns, New Orleans, aged seventy-three years, died, May 19th, of angina pectoris.

Dr. Alexander Brown Dancy, Jackson, Tenn., aged fifty-four years, died, June 10th, of heart disease.

Dr. George Francis Suker, Chicago, Ill., aged sixty-four years, died July 2nd.

Societies

At the eleventh annual meeting of the Eye, Ear, Nose and Throat Section of the Connecticut State Medical Society on May 25th, the following officers were elected: chairman, Dr. Edward J. Whalen, Hartford; vice-chair-

man, Dr. Arthur M. Yudkin, New Haven; secretary-treasurer, Dr. Walter L. Hogan, Hartford. The scientific program included motion picture films of various operations. The autumn meeting will be held in Norwich on September 28th.

Dr. W. Gordon M. Byers, Montreal, was installed president of the American Ophthalmological Society at its annual meeting, May 29th, and Dr. Walter B. Lancaster, Boston, was named president-elect. Dr. John Milton Griscom, Philadelphia, was reelected secretary. The next annual session will be held at Lucerne-in-Quebec, Canada, July 9-11, 1934. Just why another city in Canada should be chosen for our next meeting, so soon after Quebec, is hard to understand. The meeting is set for July 9, the hottest time of the year. It may be cool up there but getting there on the train is going to be terrible. Furthermore, it is a long, expensive trip when we are all broke. Why not reconsider and select a more central place for the meeting?

The Section on Ophthalmology of the American Medical Association at the recent meeting in Milwaukee elected the following officers: chairman, Dr. W. C. Finnoff, Denver; vice chairman, Dr. Frank E. Burch, St. Paul; secretary, Dr. Parker Heath, Detroit; delegate, Dr. George F. Suker, Chicago; alternate, Dr. Emory Hill, Richmond, Va.; member of American Board for Ophthalmic Examinations, to fill the three-year term expired of Dr. William H. Crisp, Dr. E. C. Ellett, Memphis, Tenn.; to fill two vacancies on the American Committee on Optics and Visual Physiology, Drs. Walter B. Lancaster, Boston, and William H. Luedde, Saint Louis. The Executive Committee recommended the appointment of a committee to consider standards for ophthalmic appliances and drugs, and to make recommendations, this committee to consist of Drs. Francis H. Adler, Philadelphia; Clifford B. Walker, Los Angeles; and Sanford R. Gifford, Chicago, chairman. A committee was also appointed to collect portraits, letters and other documents concerning the early ophthalmology of this country, the material to be placed in the Army Medical Library, Washington, D.C. Members of the committee are Drs. Harry Friedenwald, Baltimore; Arnold Knapp, New York; and Burton Chance, Philadelphia. They ask the co-operation of all ophthalmologists in the undertaking. Inquiries will be answered by Dr. Friedenwald, 1212 Eutaw Place, Baltimore.

The regular meeting of the Montana Academy of Ophthalmology and Oto-Laryngology was held, July 11th, at Anaconda, Montana. Drs. Howard Naffziger, San Francisco, and George Swift, Seattle, presented the subjects, "Neuro-Otology" and "Neuro-Ophthalmology," respectively. Dr. Roy V. Morledge, Billings, gave a report on the sectional meeting of the American Medical Association in Milwaukee, and Dr. A. W. Morse reported on the meeting of the Pacific Coast Oto-Ophthalmological Society in San Francisco where he read a paper on "The use of the electric cautery in corneal ulcers." Dr. L. G. Dunlap, Anaconda, presented a movie of "Plastic procedures" at the meeting.

Miscellaneous

The National Society for the Prevention of Blindness observed its twenty-fifth anniversary on May 24th, with "open house" at its offices.

The State Farm Life Insurance Company of Bloomington, Ill., is issuing a policy providing monthly indemnity for total blindness from whatever cause.

Personals

Dr. Edward H. Cary, Dallas, Texas, gave a radio talk on "Saving your eyes," on June 21st, at the Century of Progress.

Dr. McCluney Radcliffe, Philadelphia, was honored by his Alma Mater, Lafayette College, June 9th, with the degree of Doctor of Science.

Dr. Irving Francis Barnett has recently been appointed clinical instructor in ophthalmology at Loyola University School of Medicine, Chicago.

Dr. Samuel B. Marks, Lexington, Ky., was elected president of the Eye and Ear Section of the Kentucky State Medical Association at its last annual meeting.

Dr. J. W. Jervey, Greenville, S.C., was elected president of the American Laryngological, Rhinological and Otological Society at its recent meeting in Chicago.

A dinner was given to Dr. George H. Meeker, dean of the Graduate School of Medicine, University of Pennsylvania, June 1st, at which Dr. deSchweinitz presided.

Dr. and Mrs. Allen Greenwood have just returned from a six weeks' trip to Italy. They are spending July and August at their summer home on Cape Cod and will return to Boston early in September.

Dr. William H. Wilmer, Baltimore, received, March 25th, the decoration of the Angelo Secchi Academy of Science from Georgetown University, Washington, D.C., where, for nineteen years, he was a member of the faculty.

Dr. W. Ivan Lillie, of the Mayo Clinic, has been appointed to the Chair of Ophthalmology, Temple University. He will assume his duties at that post September 1st.